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THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 35 Number 1

January 15, 1951

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
Volume 35 — January 15, 1951 — Number 1

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POTATO ROT NEMATODE SURVEYS: 1946-1950

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H. J. Conkle and C. H. Rothe

Introduction

The presence in the United States of a nematode now known as the potato rot nematode, Ditylenchus destructor Thorne, was reported in 1943 by Earle C. Blodgett, [1] then engaged in plant-disease prevention activities for the Division of Mycology and Disease Survey of the Bureau of Plant Industry, Soils, and Agricultural Engineering. He found this nematode in potatoes from fields in the vicinity of Aberdeen, Idaho.

Apparently the same nematode had been observed in Europe as early as 1888, causing rot symptoms on potato tubers in Germany where it was considered a special strain of the widely known bulb and stem nematode, Ditylenchus dipsaci Kühn. The tuber-rot form occurs in many countries of northern Europe, where it has been a troublesome potato pest. Studies in Idaho by Mr. Gerald Thorne led him, in 1945, to consider this nematode as a distinct species, which he named Ditylenchus destructor. The only other plant established as a host in Idaho is the common dandelion, Taraxacum officinale Weber.

Occurrence in Canada

Late in 1945 the Division of Plant Protection of the Canadian Department of Agriculture announced the finding of this nematode on several farms in the seed-producing area of Prince Edward Island. From this well-known seed-potato center large shipments of certified seed are made each year to many of the Eastern States, and at the time of the discovery many carloads from the 1945 crop had already been delivered in these States.

The Bureau of Entomology and Plant Quarantine was notified promptly and furnished information on the car numbers and certificate numbers covering seed potatoes from infested fields. This information was transmitted to officials of the destination States early in 1946. As a result some seed potatoes shipped into New York State were found to be infected and were destroyed. No other infected potatoes were found in any of the States to which such seed potatoes had been shipped.

An intensive survey was immediately carried out by the Canadian authorities, and in due time assurance was obtained that all Prince Edward Island fields were free from potato rot nematode, except 10 fields comprising about 60 acres on 6 farms. These farms and adjacent areas were placed under quarantine and barred from potato production.

Occurrence in Idaho

Most of the findings up to 1949 in any one field in Idaho involved few infected tubers, and almost negligible losses. In 1949 the planting of potatoes in test plots on known infested soil at Aberdeen resulted in infections up to 75 percent in localized areas in the plots. Several theories have been advanced as to the possible origin of this nematode at Aberdeen, but as yet without confirmation.

The infested area at Aberdeen now involves 383 acres on 25 farms in Bingham County and 12 acres on 1 farm in adjacent Power County -- a total of 395 acres on which the nematode has been found. This is an increase of 55 acres and 4 farms since 1948. These farms are near or at the center of the infested area and therefore do not extend the previous limits of the infested area. The center of infestation is located approximately 1 mile southwest of the town of Aberdeen, and comprises an area approximately 20 miles long north and south, and 3 to 6 miles wide east and west.

Control measures were adopted by the Idaho Department of Agriculture in cooperation with potato growers in the infested area, following the initial finding of the pest. These measures are supervised and enforced jointly by the State Department of Agriculture and a committee of local growers. Vigilant inspections are made at grading centers throughout the infested and adjacent areas by State inspectors. Potatoes going into storage from each farm in these areas are required to be stored in separate bins. When an infestation is found, all potatoes from the infested farm are placed under quarantine and moved to the State-owned cellar at Aberdeen, where they are washed, graded, and sacked. From there the potatoes are permitted to move by sealed truck

[1] Blodgett, Earle C. Stem nematode on potato: a new potato disease in Idaho. Plant Dis. Reptr. 27: 658-659. 1943.

to dehydrators and chip factories, or they are disposed of within the quarantined area, under safeguard. Sacks are returned for fumigation under State supervision. All land on which infected potatoes have been found, except State experimental plots, is held out of potato production and planted to grain and hay crops. No seed potatoes are certified for movement from this area.

Symptoms of Damage

As observed in Idaho, the first seasonal symptoms of the potato rot nematode in potatoes are small, pinhead spots with depressed margins. As the nematode colony increases and works outward, the lesions enlarge until a considerable area, or finally the entire tuber, may be affected. At harvest time the lesions are still small. Under favorable conditions the rot lesions continue to develop in storage. Therefore, the best time for making surveys for this pest is after the potatoes have been in storage for several months.

Method of Survey

Potatoes were examined at all places where commercial lots could be found still in storage, or at dealers' warehouses during grading operations, where large quantities from each grower's lot could be observed as they passed over the grading machines. These machines at dealers' warehouses usually include one or two wide belts onto which the potatoes are dumped from a conveyer leading from the washer or directly from the hopper into which the potatoes are dumped from trucks. The belts are long enough to permit three or more graders or sorters to stand beside them. The graders pick out No. 2 and cull-grade potatoes which are usually dropped into chutes leading to other belts. The No. 2 grade potatoes go into bags for shipping, and the culls go either into bags or into large raised hoppers with bottom doors under which trucks may be driven. The culls may be hauled to starch factories or returned to farms for stock feed. The grading machines usually include an "eliminator," which permits the small potatoes to drop out into the other chutes before sorting. These small potatoes are either dumped with the culls or saved for seed. The trash, soil, and debris which accumulate around the machines are cleaned up, and either dumped outside the warehouses or hauled away to other dumping places or back to the farms.

As the potatoes pass over the grading machines, they are spread out on the belts under strong lights before the inspectors; thus every decayed spot or blemish may be observed. Tubers exhibiting symptoms of disease that cannot immediately be determined as resulting from organisms other than potato rot nematode may be picked out and examined more closely. Washing of potatoes materially increases the effectiveness of this method of examination as the soil is thus removed from them, and the moisture covering the tubers accentuates imperfections.

Approximately one carload (300 sacks) of potatoes may be run over the average grading machine in two hours. It is therefore possible for an inspector to see practically all of 100 sacks, or approximately 170 bushels, in half an hour. By making examinations at each grader several times during the day, different parts of one or more growers' lots may be examined.

Potatoes are usually scalped, or given a preliminary sorting over portable grading machines at the cellars, to eliminate the badly rotted or decayed tubers before they are sacked for transportation to dealers' warehouses for grading. Partially rotted potatoes are not eliminated at this operation as portions of many may be saved for No. 2's in the final grading. Since any lot of potatoes infected with potato rot nematode will include many tubers with partially developed rot lesions, major attention is given to tubers exhibiting partial breakdown rather than to the badly or completely decomposed tubers. Complete breakdown may be due to other organisms. Rot nematode damage in completely rotted tubers may be obscured by decay caused by other organisms. Therefore, the potatoes thrown out in the scalping operations do not materially reduce the chances of detecting this pest if present in a lot.

Surveys in Eastern United States

During the summer of 1946 an extensive survey of potato plantings and of potatoes at harvest time was conducted by the Bureau of Entomology and Plant Quarantine in cooperation with the States of Connecticut, Delaware, Maine, Maryland, Michigan, Minnesota, New Hampshire, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Rhode Island, Vermont, Virginia, and Wisconsin to determine whether potato rot nematode infestation centers were present in any of these States. The survey, which was continued through the storage period of 1946-47 by State personnel, was made in 94 counties of 16 States, and potatoes were examined at 2,171 locations. No infestations of the potato rot nematode were found.

Observations for this pest have been continued by inspectors engaged in survey activities for the golden nematode incidental to such survey work over wide areas of the eastern United States and no potato rot nematode infection was found in many thousands of bushels washed, or in 1,269 potato storage houses examined in Long Island, other sections of New York State, and other eastern States.

Surveys in Western United States

Since injury resulting from this nematode is similar to that produced by certain other potato diseases, it was recognized from the outset that undetected centers of infestation might be established in other potato areas in Idaho or elsewhere in the United States.

In view of the finding in 1948 of an additional area of infestation in Power County, Idaho, and because of the interest in this problem developing in other States, Idaho authorities requested a conference with Bureau officials in December 1948 to consider the potato rot nematode situation. Facts brought out at the conference suggested the desirability of a more comprehensive survey throughout the Western States, including Idaho. Such a survey was approved by members of the Western and National Plant Boards. Plans for the survey were worked out in cooperation with the Bureau of Plant Industry, Soils, and Agricultural Engineering and officials of the States to be surveyed.

Table 1. Summary of inspections for potato rot nematode, by States.

State	1949				1950			
	Number of counties inspected	Potatoes inspected (cwt.)	Potato specimens collected: Number	Potato specimens collected: Counties	Number of counties inspected	Potatoes inspected (cwt.)	Potato specimens collected: Number	Potato specimens collected: Counties
California	7	2,603	5	3	8	11,963	36	7
Colorado	8	3,066	8	4	15	22,190	70	14
Idaho	15	77,348	110	13	27	95,759	222	24
Montana	12	4,330	24	11	9	1,316	9	5
Nebraska	-	-	-	-	5	2,518	2	1
Nevada	3	140	2	2	4	1,330	-	-
Oregon	5	40,758	14	3	6	23,570	25	4
Utah	10	585	3	2	11	9,650	30	11
Washington	6	4,155	14	6	8	14,264	2	2
Wyoming	-	-	-	-	7	1,660	-	-
Totals	66	132,985	180	44	100	184,220	396	68

The 1949 survey was begun March 12 and completed May 7, and covered important potato-growing areas of California, Colorado, Idaho, Montana, Nevada, Oregon, Utah, and Washington. In some localities it was found that, owing to the lateness of the season, potatoes had already been moved to market. An adequate survey was therefore not possible in such localities.

In view of the limitations of the 1949 survey, additional inspections were deemed desirable in order to cover areas previously missed, and to reexamine storages in which only a limited sampling could be done in the brief period devoted to the survey in the spring of 1949. Consequently, a further cooperative survey was made from January 17 to April 10, in the same States and, in addition, in Nebraska and Wyoming.

In connection with the 1949 and 1950 surveys, soil samples were collected in potato cellars and at graders, and sent to laboratories of the Division of Golden Nematode Control for examination for the presence of golden nematode. In the 1949 survey 378 soil samples were collected in 62 counties, and in the 1950 survey, 9,768 samples in 106 counties.

Samples of potatoes collected during the survey which were suspected of being infected with potato rot nematode were sent for taxonomic examination to nematologists of the Bureau of Plant Industry, Soils, and Agricultural Engineering, except for those collected in Idaho, which were sent to the Idaho Pest Control Commission laboratory at Aberdeen, where preliminary examination of them was made.

Before the surveys were started all inspectors of the Bureau of Entomology and Plant Quarantine were trained at the Aberdeen Branch of the Idaho Agricultural Experiment Station in detecting symptoms of potato rot nematode damage.

Summaries of Surveys by States

CALIFORNIA: -- The surveys in California were made in cooperation with the California Bureau of Plant Pathology and with the assistance of county agricultural commissioners and State and county inspectors, and included inspections in nine counties, -- Kern, Madera, Modoc, Plumas, San Benito, San Joaquin, Siskiyou, Sonoma, and Tulare. The largest storage area in the State is in Modoc and Siskiyou Counties in the vicinity of Tulelake. This area lies just south of the Oregon-California State line and directly south of and adjoining the Klamath Falls potato area in Oregon.

COLORADO: -- The Colorado surveys were made in cooperation with the Colorado State Entomologist and with the assistance of State inspectors in the 15 counties of Alamosa, Conejos, Costilla, Delta, Eagle, Garfield, Larimer, Mesa, Montrose, Morgan, Pitkin, Rio Grande, Saguache, Sedgwick, and Weld. Most of the potatoes were inspected at commercial graders which handle the bulk of potatoes in the State, and which were operating steadily throughout the survey periods. Each county was visited several times during each survey, resulting in a good representative coverage of potato plantings from different parts of each county.

IDAHO: -- The major part of the surveys was concentrated in Idaho because of the location at Aberdeen of the only definitely known infestation of the potato rot nematode in this country, and because potato production is a major industry in many parts of the State. They were made in cooperation with the Idaho State Department of Agriculture and the Idaho Pest Control Commission, with the assistance of Federal-State grade inspectors, county agents, and others. In 1949 inspections were made in 15 counties, and in 1950 potatoes in 27 counties were inspected.

According to estimates of the Production and Marketing Administration of the United States Department of Agriculture, potatoes were grown on 148,734 acres in Idaho in 1948, 91 percent of which were in the 15 counties in which the 1949 survey was conducted. Eighty percent of the certified or registered seed produced in Idaho was also grown in these 15 counties. The greater part of this acreage is on irrigated land in the Snake River Valley from Fremont County on the northeast to Twin Falls and Gooding Counties on the southwest. It is divided roughly into two areas -- viz, the Twin Falls-Burley area comprising the six counties of Cassia, Gooding, Jerome, Lincoln, Minidoka, and Twin Falls, and the Upper Valley area comprising the five counties of Bingham, Bonneville, Fremont, Jefferson, and Madison.

Between these areas is a narrow strip of irrigated land between the Snake River lava plains and the Snake River at the American Falls Reservoir, centering at Aberdeen and extending into Bingham and Power Counties. All the infested farms in Idaho are in this area. The State of Idaho has annually conducted intensive potato examinations at all potato grading centers in the Aberdeen area from harvest time in the fall throughout the shipping season. For this reason it was deemed unnecessary to expand additional effort in this thoroughly surveyed area. The cooperative Federal-State activities were therefore devoted primarily to the Twin Falls-Burley and Upper Valley areas, extending into northern Bannock County, and the seed-producing areas in the vicinity of Ashton, Fremont County; the Teton Basin, Teton County; Salmon, Lemhi County; and Darlington, Butte County.

At the time of the 1949 survey in Idaho, few potatoes remained in storage in the smaller producing sections of the State. However, the 1950 survey was begun about two months earlier, and inspections were extended into northern Idaho from Lewis County north to the Canadian border, in which area the greater part of the potatoes are grown as certified seed. A number of inspections were also made in parts of western and southeastern Idaho, which were not covered in the 1949 survey. Excellent coverages were obtained in all seed-producing sections; in fact, as much as 99 percent of the growers' lots were inspected in Boundary County.

Examinations in both years were made primarily at dealers' warehouses while potatoes were being graded. This afforded the best examination material and the best opportunity to observe large quantities of potatoes from each growers' lot.

Most of the potatoes in the Upper Valley area were washed before grading. This added considerably to the ease of examination. Examination of stored potatoes -- particularly seed potatoes -- was also made in a number of potato cellars. In many places it was found that most of the seed potatoes had been graded and tagged before arrival of the inspectors, but cull piles were

usually available for examination. The 15 counties in which inspections were made in 1949 are: Bannock, Bingham, Bonneville, Butte, Cassia, Fremont, Gooding, Jefferson, Jerome, Lemhi, Lincoln, Madison, Minidoka, Teton, and Twin Falls. In 1950, inspections were made in all of these counties and also in the 12 counties of Ada, Bonner, Boundary, Canyon, Caribou, Custer, Idaho, Kootenai, Latah, Lewis, Owyhee, and Payette.

MONTANA: -- In Montana, in cooperation with the Division of Horticulture, State Department of Agriculture, Labor, and Industry, and with the assistance of State inspectors, surveys were made in the 12 counties of Beaverhead, Broadwater, Deer Lodge, Flathead, Gallatin, Jefferson, Lake, Lewis and Clark, Madison, Missoula, Powell, and Ravalli. These counties produced 72 percent of the State's potato crop in 1949.

The 1950 survey in Montana was shifted somewhat to take in parts of the eastern section of the State where inspections were not made in 1949, thus adding three new counties -- Dawson, Richland, and Yellowstone -- to the surveyed area. Resurveys were made in six of the counties covered the previous year.

In this State most of the inspections had to be made in cellars as the potato areas are widely scattered and it was seldom possible for the inspector to be on hand when potatoes were being graded.

NEBRASKA: -- No survey was made in Nebraska in 1949, but in 1950 the survey was extended to include inspections in the important potato-growing area of the western part of the State. In cooperation with the Nebraska State Entomologist inspections were made in the counties of Box Butte, Cheyenne, Kimball, Morrill, and Scotts Bluff. Approximately 68 percent of the potatoes grown in Nebraska are produced in the western part of the State, a large portion of which are washed, graded, and sacked for shipment in Scottsbluff.

NEVADA: -- Surveys in Nevada were made in cooperation with Division of Plant Industry, Nevada State Department of Agriculture, with the assistance of State inspectors. The 1949 survey was made from March 18 to 23 in Lyon, Pershing, and Washoe Counties where approximately 86 percent of the State's potatoes are produced. In 1950, the same area was surveyed about a month earlier, and a small potato area in Elko County was included.

OREGON: -- The main potato-growing areas in Oregon are in the vicinities of Ontario, Redmond, and Klamath Falls, in which inspections were made during both the 1949 and 1950 surveys in cooperation with the Division of Plant Industry, Oregon State Department of Agriculture, and with the assistance of State inspectors. In the Redmond and Klamath Falls areas an excellent coverage was obtained during both surveys as potato-grading operations were under way during the entire time inspectors were in those areas. All lots of potatoes found remaining in the areas were inspected either at graders or at storage locations. The Klamath Falls area joins a similar large potato-producing area in Modoc and Siskiyou Counties, California.

Surveys were made in Baker, Crook, Deschutes, Jefferson, Klamath, and Malheur Counties.

UTAH: -- Potatoes in the ten Utah counties of Beaver, Box Elder, Cache, Garfield, Iron, Piute, Salt Lake, Utah, Washington, and Weber were inspected in 1949. Reinspections were made in 1950 in nine of these counties and in two additional counties -- viz., Davis and Sevier. The surveys were made in cooperation with the State Department of Agriculture and with the assistance of State inspectors. Most of the inspections in the State were made of potatoes in storage as at the time inspections were made graders were in operation at only a few places.

WASHINGTON: -- The office of the Supervisor of Horticulture and State inspectors cooperated in surveys in this State, which were made in the six counties of Grant, Kittitas, Skagit, Snohomish, Whatcom, and Yakima in 1949. Reinspections were made in these counties in 1950 and also in the counties of Benton and Clark. Since most of the Washington-grown potatoes are moved to market immediately after harvest, only a few potatoes could be found in storage at the time the surveys were made; however, representative inspections were possible in all the principal potato-growing areas of the State.

During the 1950 survey in this State, potato specimens from one location near Mount Vernon, Skagit County, were found with symptoms resembling potato rot nematode damage. Upon examination of these specimens by nematologists, nematodes were found that were suspected to be Ditylenchus destructor Thorne.

WYOMING: -- Surveys in this State, conducted in cooperation with the office of the State Entomologist and with the assistance of State inspectors, were made only in 1950. Inspections were made in seven counties -- Fremont, Goshen, Johnson, Laramie, Park, Platte, and Sheridan -- which produce about 86 percent of the potatoes grown in the State.

Potato acreage in Wyoming is not large, and is largely confined to the Powell, Riverton, Torrington, and Glendo areas. Seed potatoes are grown at Sheridan, Buffalo, Glendo, and Lingle. About two-thirds of the crop remained in storage at the time of the survey, thus affording an excellent opportunity for inspection of potatoes from all the potato-growing areas.

Summary

The potato rot nematode surveys offer reasonable assurance as to the distribution of this pest in the Western States. None was found in two years' survey in California, Colorado, Montana, Nevada, Oregon, or Utah; nor was any found in the 1950 survey in Nebraska or Wyoming. In Idaho, State inspectors found only a few additional infested fields, which were close to previously known infestations. The finding of suspect specimens at Mt. Vernon, Washington, suggests consideration of the advisability of making further surveys in that State, and perhaps in other western States.

Representative examinations were made of potatoes in all the principal late potato-producing areas of ten States, and it was found that a small staff of inspectors could make a very thorough and extensive coverage by examining potatoes as they are run over grading machines preparatory to sacking for shipment, especially in those places where potatoes are washed before grading.

DIVISION OF DOMESTIC PLANT QUARANTINES, BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE

ROOT-KNOT NEMATODE ON CANAIGRE

Harold W. Reynolds and Bailey Sleeth

The fleshy roots of canaigre, Rumex hymenosepalus Torr., a plant native to the arid areas in Arizona, New Mexico, adjoining States and Mexico, are high in tannin, starch and sugar. Canaigre is a potential farm crop in the Southwest, because of its high tannin content, which may vary from 10 to 35 percent on a dry weight basis,[1] and the vital importance of tannin in our national economy. During the past two years an experimental planting of several acres of canaigre has been grown on the U. S. Department of Agriculture Yuma Mesa experimental area at Yuma, Arizona by the Division of Tobacco, Medicinal and Special Crops, mainly for developing agronomic information on how to grow canaigre as a crop and for securing sufficiently large number of roots for pilot plant tests.

FIGURE 1. Root-knot nematode infection on canaigre roots. The fleshy root on the left shows numerous warty, elevated lesions while the smaller rootlets on the remaining three roots show typical galling.

During the summer of 1950, at the time the canaigre roots were being harvested, infection caused by a root-knot nematode, Meloidogyne incognita (Kofoid & White) Chitwood, was observed on both the small roots and large fleshy roots as shown in Figure 1. The roots showed pronounced galling typical of root-knot nematode damage to many crops such as tomato, grape, and okra. The injury on the fleshy roots gave a warty appearance with slight elevations typical of root-knot infection on other fleshy subterranean stems and roots, such as Irish potatoes and sweet potatoes. An examination of these roots revealed an abundance of adult female root-knot nematodes embedded 1 to 4 mm. beneath the surface. In many cases, especially with the more shallowly embedded parasites, large egg masses containing an abundance of eggs had extruded to the surface of the roots.

It is not known to what extent root-knot nematodes will reduce yields or impair the roots for processing. However, if canaigre becomes a stable farm crop and is planted in considerable acreage, it would be well to keep in mind that root-knot nematodes could readily be disseminated if infected roots or root pieces were used in propagation. Also, the plant is well adapted to sandy or coarse textured soils, and it is in these soils, once infested, that root-knot nematode populations rapidly increase and soon become a serious hazard to the growing of susceptible crops.

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[1]Russel, G. A., J. S. Rogers and E. C. Stevenson. Canaigre Investigations, Jour. Am. Leather Chem. Assoc. 39: 479-491. 1944.

VERTICILLIUM WILT SPREAD TO DISEASE-FREE SOIL BY INFECTED COTTON STALKS

Lester M. Blank and Philip J. Leyendecker, Jr.

The factors contributing to the spread of Verticillium wilt of cotton in the irrigated regions of the Southwest have been in question for a number of years. Rudolph and Harrison (1) have shown conclusively that the pathogen is not carried within the seed.

It has been demonstrated (2) that in southern New Mexico the causal agent remains viable in dead infected cotton stalks left in the field throughout the winter. Isolations of the fungus were made from the stem and larger branches as late as June 1 following a minimum winter temperature of -5° F. These findings indicate that Verticillium could be established in the soil from infected stalks. If this is the case, the diseased stalks would be a factor in the spread of the disease, since they are mechanically cut, or chopped, and plowed under before seedbeds are prepared in this region. The following experiment was designed to determine the validity of this reasoning.

Ten plots (10 feet x 90 feet) were laid out on a Gila fine sandy loam which had not been planted to cotton in 25 years and was assumed to be free of the wilt organism. In late October 1949, five lots of 100 dead wilt-infected cotton stalks were pulled from a field and stored out-of-doors until used. Each 100-stalk sample was cut into 4 to 6-inch pieces and placed on the surface of designated plots on December 1, January 1, February 1, March 1, and April 1. Immediately following the application of the infected stalk material the treated plot and an adjacent untreated plot were plowed. All ten plots were bedded in early April, irrigated, and planted on April 20 with acid-delinted seed of Texacala variety of cotton. This seed had been produced in an area free from Verticillium wilt, thus obviating question as to carryover of the wilt organism in or on the seed. Normal farming operations were followed throughout the growing season.

Leaf symptoms of Verticillium wilt were observed in the treated plots early in July, and disease counts were made in all plots on August 9, August 31, and September 25. The results of the final disease record are presented in the accompanying table.

Date of treatment	Percentage of plants showing wilt symptoms	
	Treated plots: infected stalks applied	Untreated plots: no stalks
December 1	99	0
January 1	98	0
February 1	97	0
March 1	95	0
April 1	83	2

The final percentages of wilted plants on treated plots show little difference between the five dates of treatment, although the earlier counts gave greater differences in the disease values. It will be noted that the April check plot contained a few diseased plants, probably because of the accidental transfer of infected stalk material from the treated plot during plowing or subsequent cultural operations. The other four check plots remained free from wilt throughout the season.

Yield data were obtained on equivalent areas in the ten plots. Calculated on the basis of pounds of seed cotton per acre the wilt-infested plots yielded at the rate of 1637 pounds per acre and the check plots at 2686. The reduction in yield due to wilt amounted to 1049 pounds of seed cotton per acre, or 398 pounds of lint cotton. To the writers' knowledge these data furnish the first accurate measurement of the effect of Verticillium wilt upon yield of cotton.

These data demonstrate the potentialities of wilt-infected stalks as a means of spreading wilt to areas previoulsly free of the disease, inasmuch as practically all of the plants became infected on plots where the diseased stalks were applied and turned under. It seems highly probable that the current practice of turning under of cotton stalks has contributed to the build-up of disease inoculum in the soil. The question of the value of removing and destroying diseased stalks as a control measure must remain unanswered until additional experimental work can be undertaken. It is recognized that, in severely infested fields, the destruction of diseased stalks through burning or some such procedure can remove only a part of the potential inoculum and that a considerable amount of infective material will remain in the lower roots and in the soil. Stalk disposal would probably be of value in fields showing only occasional diseased plants. It is suggested that, under such conditions, the removal of diseased plants during the growing season, while symptoms are evident, might contribute much to a reduction in spread of the disease throughout the remainder of the field during subsequent seasons.

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DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE AND THE DEPARTMENT OF BOTANY OF THE NEW MEXICO AGRICULTURAL EXPERIMENT STATION, STATE COLLEGE, NEW MEXICO. CONTRIBUTION NO. 61

COTTON SEEDS ARE CAPABLE OF CARRYING VERTICILLIUM

Ross M. Allen

For some years there has been conjecture as to the possibility of transmission of Verticillium wilt of cotton by naturally infected seeds. A number of research workers have expressed the opinion that seeds of cotton have been to some extent responsible for the unsatisfactorily explained spread of the disease to areas from which the disease had never previously been reported. It appeared reasonable to Barducci and Rada (1) that the extended spread of the fungus through the entire plant of cotton, and particularly in the young tissues, might involve fungus hyphae reaching the seeds, and that this might be one of the means of spread of the disease.

FIGURE 1. (a) Seeds of SxP variety showing Verticillium colonies growing from needle puncture holes and from micropylar openings. Inoculations were made Mar. 8, 1950. Seeds photographed Nov. 15, 1950; (b) at left, a seedling of SxP variety from a seed hypodermically inoculated April 5, 1950. At right, a seedling of 1517 RB variety similarly inoculated April 14, 1950. Verticillium growth appears on cotyledons of both seedlings and has spread to the agar of both plants; (c) seedling of 1517 RB variety from a seed inoculated April 14, 1950. Circular, white colonies of the fungus appear on the cotyledons.

Kadow (2) proved that Verticillium is carried in the seeds of eggplant and tomato. Taubenhaus (4, 5) reported that 8.3 percent of 1440 cotton seeds tested in 1936 contained Verticillium, and that in the following year 2.3 percent of 1600 seeds gave positive results. On the other hand,

Rudolph and Harrison (3) expressed the opinion that cotton seeds were relatively unimportant in the dissemination of Verticillium wilt in California following extensive testing of seeds taken from infected plants. These writers did not state that infection of cotton seed is impossible but that in view of accumulated data that it is improbable. The statement was also made "that there is no assurance that the parasite having reached an occasional seed would remain alive there and attack the seedling the following spring. Such parasitized seed might not even germinate." My report concerns the statement quoted above.

Although it is not yet definitely known that Verticillium wilt can be or is transmitted by means of naturally infected seeds taken from infected plants, it is definitely known that seeds inoculated with the fungus are capable of carrying the latter over extended periods of time, in the interior of the seeds, and still germinate.

Cotton seeds of SxP variety were inoculated March 8, 1950, under aseptic conditions with a pure culture of Verticillium albo-atrum Reinke & Berthold, by hypodermic injection of inoculum containing spores and portions of the fungus hyphae. Seeds were soaked in sterile distilled water to soften the seed coat and thus facilitate penetration of this part by the inoculating needle. After soaking for a short period the seeds were transferred to a 1:1000 solution of mercury bichloride for surface sterilization for three minutes and then rinsed in distilled water. After inoculation, this lot of seeds was placed in a sterile, covered dish and allowed to dry. On March 10, white fungus growth identified as Verticillium was observed at the puncture hole in many of the seeds as well as at the micropylar opening of others. About ten days later these colonies ceased active growth. The seeds (Fig. 1) were kept in the dish at room temperature until October 28, 1950, when some of them were cultured on potato-dextrose-agar following a five-minute immersion in Rada's (1) disinfectant of alcohol and mercury bichloride. After seven days on the agar at 25° C. the fungus was again produced from within the seeds.

In other, but similarly conducted experiments, cotton seeds of several varieties including SP, Paula C, Pima 32, and 1517 RB were inoculated. These seeds were stored in glass flasks stoppered with cotton plugs and kept in the laboratory at room temperature, thus somewhat approximating normal storage conditions of cotton seeds prior to planting. Of approximately 200 of these seeds cultured on agar, half of them, representing all inoculated varieties, produced Verticillium as shown in Fig. 1 (b). Very few seeds failed to germinate at least in part, and approximately 75 percent of them reached the cotyledon stage (Fig. 1, c) in spite of the fluctuating storage conditions and the injury inflicted by the inoculating needle.

In several instances it has been found that the fungus can be cultured from inoculated seeds as long as eight months after inoculation. It is evident that cotton seeds can carry Verticillium albo-atrum in their interior if the fungus reaches them by natural infection.

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DEPARTMENT OF PLANT PATHOLOGY, AGRICULTURAL EXPERIMENT STATION, TUCSON, ARIZONA

ON THE OCCURRENCE OF COLLETOTRICHUM DERRIDIS IN TAIWAN

Lee Ling

Very recently van Hoof[1] described a new species of Colletotrichum, C. derridis, as the cause of leaf blight and die-back disease of Derris in Java and Celebes. In Taiwan (Formosa), South China, this disease has been observed since 1940. During the period from 1934 to 1939, over half a million cuttings of Derris elliptica (Wall.) Benth. were imported from Malaya into Taiwan and were distributed over the island for propagation. It is suspected that the disease was introduced into Taiwan with these cuttings. In October, 1941, a survey made by the Taiwan Agricultural Research Institute revealed that this disease had become established in the island wherever D. elliptica was grown, causing heavy damage especially in nurseries in Taichung and Hwalienkong. Although no attempt was made to name the pathogen specifically, its morphology and physiology have received considerable study by the Division of Plant Pathology of the Institute. An abstract[2] was published in 1947.

Outside Taiwan, this disease has also been reported from Hainan Island.[3]

The Disease

The disease occurs on leaves, petioles, and stems of both young and mature plants of Derris. The initial infection appears on the upper surface of the leaf as minute, brownish specks. Under humid conditions, the lesions enlarge rapidly and sometimes coalesce, attaining up to 17 mm. in diameter. Such lesions are roughly circular or irregular in outline with yellow brown to deep brown centers and darker borders. The infected leaves often become distorted or malformed, but this malformation is not a constant characteristic of the disease (Fig. 1).

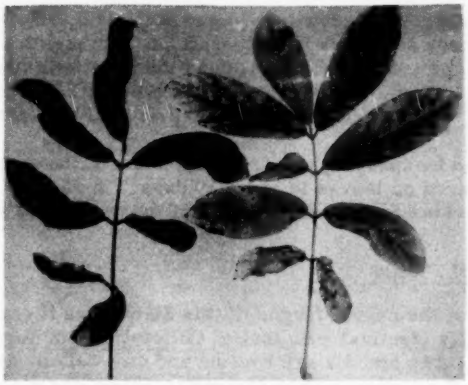

FIGURE 1. Symptoms on leaves of Derris elliptica infected by Colletotrichum derridis.

FIGURE 2. Symptoms on Derris infected by Colletotrichum derridis, showing withering of leaves as a result of infection on petioles.

On petioles, the lesions are elongate-elliptical and dark brown. With age, they become dry and depressed, and turn lighter in color. When the petioles are badly infected, the leaves droop and may not recover (Fig. 2).

On stems and tendrils (Fig. 3), the infection is characterized at first by light brown, slightly sunken spots, which later develop into elongate cankers, measuring 2-15 x 1-6 mm. The centers of the cankers, in which are embedded the acervuli of the causal fungus, are brown to grayish white and surrounded by dark borders, and are sometimes longitudinally fissured. An

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[1] van Hoof, H. A. A new species of Colletotrichum (Fungi imperfecti). Bull. Bot. Gard. Buitenz. 18: 473-478. 1950.

[2] Annual report of the Taiwan Agricultural Research Institute for 1946. 136 pp. 1947. (In Chinese).

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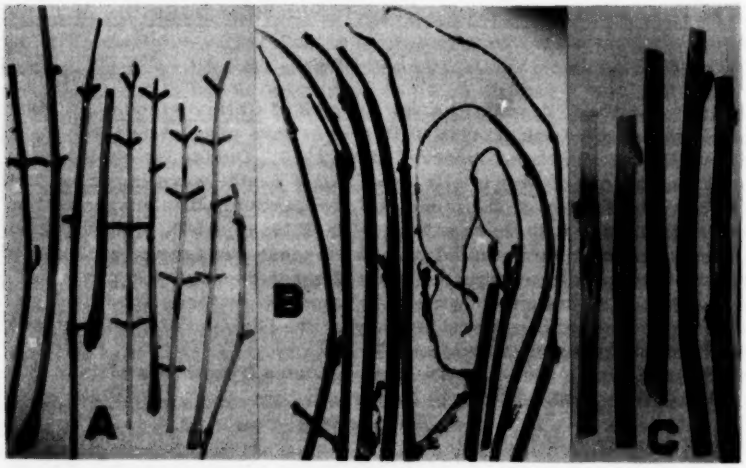

FIGURE 3. Symptoms on Derris infected by Colletotrichum derridis. A, lesions on petioles; B, die-back of upper parts of tendrils; C, lesions on stems.

infected stem, which may be partially or completely girdled by the canker, often bends over and eventually breaks off at the lesion, resulting in the death of part or all of the plant.

Surviving diseased plants are considerably stunted in growth. On one farm at Hwalienkong, both the diseased and healthy plants grown on sandy soil were random-sampled one month after transplanting in the field to compare their development under natural conditions. It was found that the stems of diseased plants averaged only 5.3 cm. in height as compared with 12.6 cm. for healthy plants. The average figures for the length of roots of diseased and healthy plants were 4.6 cm. and 16.1 cm. respectively.

When young plants of Derris were inoculated by spraying a spore suspension of the causal fungus in the usual manner, initial lesions appeared on leaves in about 10 days. In 15 to 20 days after inoculation, lesions on petioles became obvious and the upper parts of the inoculated plants usually died shortly afterward.

The Causal Fungus

FIGURE 4. Part of acervulus of Colletotrichum derridis, showing the formation of a conidium on the apex of a seta.

The morphology of the causal fungus of this disease as it occurred in Taiwan is essentially identical with that of Colletotrichum derridis described from Java. The conidia are hyaline and one-celled, and measure 12-21 x 4.5-6 microns. They are generally oblong with rounded ends, but modification in form often occurs. The setae are present both on the host and in culture, arising from among the conidiophores or at the margin of the acervulus. They are stiff, pointed, simple, brown, and 2- to 8-septate, varying from 63 microns to 174 microns in length and from 3 microns to 6 microns in width. The conidiophores are hyaline, continuous, and often branched, measuring 9-21 x 3-6 microns

As observed on the host and also in culture, the setae frequently function as conidiophores. Conidia bud out from the apices of setae in exactly the same manner as they are formed on the conidiophores. This seems to be a constant characteristic of this fungus, as was also recorded by van Hoof[4] (Fig. 4).

The conidia begin to germinate in 3 to 4 hours under favorable conditions. The optimum temperature for germination was found to be 28° C., and maximum between 35° and 40° C.

The fungus grew well on most agar media tested. On potato-dextrose agar, it produced dense mats of grayish white mycelium with conidia borne at the ends of non-differentiated, branched hyphae and

[4] loc. cit.

setae as well as in acervuli. The temperature requirements for mycelial development appeared to be same as that for spore germination. Mycelial growth on small agar blocks was killed by an exposure of 15 minutes at 50° C., or of 5 minutes at 53° C.

Control Measures

Selection of disease-free cuttings, coupled with field sanitation, offers the best possibilities for the control of this disease.

Derris, for commercial purposes, is grown only from cuttings, which are first planted in nursery beds and are then transplanted to the field when rooted. Apparently the primary source of inoculum of this disease is chiefly the pathogen carried by cuttings. Selecting cuttings only from disease-free mature plants eliminates largely the initial source of infection. Early applications of Bordeaux mixture, either in the form of 2-2-50 or 4-4-50, were found to be effective in preventing epidemic development of the disease but are not practical in the field under local conditions.

The pathogen may also persist for some time on plant residues. The removal and burning of infected plants and debris reduce sources of secondary inoculum.

Derris chinensis Benth. was found to be highly resistant to this disease, but does not contain active principles sufficiently high to be of commercial importance as an insecticide.

UNITED NATIONS FOOD AND AGRICULTURE ORGANIZATION

PHYTOPHTHORA PARASITICA DISEASES OF CINCHONA IN CENTRAL AMERICAN FIELD PLANTINGS[1]

Michael Szkolnik

This paper describes certain disease problems occurring in field plantings of cinchona[2] in Guatemalan and Costa Rican plantations. At these plantations the losses of cinchona trees from infections by Phytophthora parasitica Dast. has been exceptionally heavy during the wet season each year. Most of the investigations were made at Finca El Naranjo, a cinchona plantation located approximately between the 4000 and 7000 ft. elevation on the southwestern slope of Volcano Santa Clara in Guatemala. Finca Los Ensayos, the plantation in Costa Rica which suffers similar disease losses, is located at an elevation range of approximately 2500 and 3800 feet on the northern and northeastern slope of an inland mountain range.

In the many publications covering cinchona culture in the Far East, the largest cinchona growing area in the world, and Africa, no definite reference appears to have been made tying in P. parasitica specifically with any of the many disease problems reported (5). Limited reference has been made to this fungus on cinchona grown commercially or experimentally in the western hemisphere.

In 1945 Crandall and Davis (2) described a disease of cinchona seedlings in Peru. They isolated a Phytophthora from these seedlings and tentatively identified the causal fungus as Phytophthora parasitica Dast. The fungus was reported to infect seedlings principally via the tender leaves and succulent stems causing brown necrotic lesions of indefinite margins. The infection spread down the petioles and stem causing necrosis of the latter and eventual death of the plant. Reinking (6) reported on a dieback and canker of Cinchona ledgeriana and C. calisaya. He reported that a Phytophthora sp. was isolated from diseased plants and considered the fungus to be the causal organism of the disease. Kevorkian (4) reported the presence of commonly-occurring stem cankers on young cinchona plants in Puerto Rico. This disease was later reported by Harper and Winters (3) and Winters (8) to show symptoms similar to those described by Crandall and Davis (2).

In Guatemalan and Costa Rican field plantations of cinchona, P. parasitica was found to be responsible for two dissimilar phases of disease. Although the one fungus causes the two phases, the general pattern of these is such that, under ordinary field observation, they may easily be mistaken for two unrelated diseases. To avoid ambiguity they will be treated separately in this paper under the two names which have been used to designate them in the field. The connotation "top blight" (perhaps more appropriately called "leaf and stem wilt") has been applied to the phase of the disease involving sprout and branch infections; the term "girdle canker" has been used specifically to designate trunk cankers caused by the same fungus.

SUSCEPTIBILITY

Variable degrees of susceptibility to infection by P. parasitica have been observed among different cinchona species, varieties, and hybrid clones. Cinchona ledgeriana was found to be most vulnerable to natural infections by this fungus. Hybrid clones of strong C. ledgeriana parentage are generally very susceptible. C. succirubra is highly tolerant, but not immune, to infection by P. parasitica. Despite its tolerance and its ability to grow very well on its own roots, C. succirubra is not satisfactory for commercial bark production because the bark yields a very low percentage of quinine. In commercial plantings it is successfully used as a rootstock to support good growth of hybrid clones with higher quinine content which do not grow well on their own roots and which cannot be propagated as pure line strains from seeds. A commercial hybrid clone designated as Clone ZM, a cross between C. ledgeriana and C. succirubra, shows a degree of susceptibility to P. parasitica intermediate between that of the parent species. Certain hybrid crosses with strong C. succirubra parentage show considerable tolerance to P. parasitica; however, low quinine content of their bark does not warrant growing them commercially.

Natural trunk infections of C. calisaya by P. parasitica are seldom observed at Finca El Naranjo; however, artificial inoculation of the fungus into 80 C. calisaya trunks resulted in severe infections with the complete girdling of 74 trunks within six weeks. Thus C. calisaya is not tolerant to the fungus but the bark apparently possesses some other barrier to natural infection.

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[1]Observations and experimentations were made while the author was controlling cinchona diseases for Experimental Plantations Inc., Guatemala and Costa Rica, Central America, a subsidiary of Merck and Company Inc., Rahway, New Jersey.

[2]A tropical tree, the bark of which yields the antimalarial quinine and other related alkaloids.

In 1947, B. A. Krukoff[3] submitted several diseased specimens of cinchona to C. M. Tucker for determination of the causal organism. Among these specimens were a stem of C. ledgeriana infected with "top blight" in Costa Rica and diseased trunk bark from C. ledgeriana infected with "girdle canker" in Guatemala. P. parasitica isolates No. 938 and No. 941 were cultured from the two specimens respectively. Early in 1948, P. P. Pirone inoculated these two isolates into branches of small C. ledgeriana and C. succirubra trees at the New York Botanical Garden greenhouses. Within two weeks the stems of C. ledgeriana suffered advanced infections and complete stem girdle in the region of inoculation. Stem cankers on these trees extended at least four inches above and below the site of inoculation. The same P. parasitica isolates failed to infect the inoculated C. succirubra branches.

TOP BLIGHT

"Top blight" infections of cinchona trees by P. parasitica in field plantings occur on trunk sprouts and on branches of susceptible trees (Fig. 1). The disease occurs more commonly on the sprouts growing at the base of trunks than it does on branches. Top blight also attacks young nursery stock of C. ledgeriana and susceptible hybrid cinchona clones under prolonged wet conditions.

FIGURE 1. Phytophthora parasitica disease symptoms on cinchona. A. "Top blight" wilted C. ledgeriana sprouts at base of Ledger trunk; S, designates C. succirubra portion of trunk. B. (1), healthy C. ledgeriana sprouts; (2), C. ledgeriana sprouts with early "top blight" infections. C. C. ledgeriana tree with severe "top blight" infection of lower branches. D. Sprout of Hybrid Clone Z-194 with "top blight" infection (TB) initiated on the stem.

TOP BLIGHT OF SPROUTS: Top blight of sprouts is characterized by initial coffee-brown spot infections of leaf, petiole, or stem. The number of individual spot infections is influenced by the abundance of fungus inoculum in the vicinity and the favorableness of weather conditions to infection. Under suitable conditions a single spot infection of a leaf can spread rapidly throughout the entire blade and progress down the petiole and stem. Multiple individual infections often occur at about the same time. Individual deep brown circular spots are almost equally prominent

[3]Central American Manager, Experimental Plantations Inc.

on both sides of the leaf blade. As the spots enlarge they usually lose their initial circular shape and become irregular in outline (Fig. 1, B). Growth of individual spot infections and coalescence with others result in variably sized lesions of indefinite design which continue to enlarge and infect additional healthy tissue. Leaf and petiole infections advance down the stem killing invaded tissue and leaving the sprout in a brown, wilted condition (Fig. 1, A).

Direct infection of the sprout stem well below the terminal region (Fig. 1, D) causes stem girdle and progressive infection up and down the stem. This infection may actually reach the terminal portion of the stem, but often the girdling of the stem in the region of initial infection causes the stem and foliage distal to this region to wilt before the fungus has had time to reach the terminal parts. P. parasitica apparently does not infect dead tissue nor does it remain alive long in tissue which it has infected and killed. Top blight infections of trunk sprouts, whether initiated in the leaf, petiole, or stem, often advance to the main trunk. Continued growth of the fungus here results in the trunk infection called "girdle canker" (described later).

Certain cinchona clones (e. g. Hybrid Clones Z-194 and Z-218) sprout more abundantly than others and are also very susceptible to infection by P. parasitica. These clones usually suffer the greatest number of top blight infections during the wet season. In addition to clonal sprouting characteristics, excessive production of trunk sprouts is often induced by severe branch pruning, mechanical or storm damage to trees, death of many branches due to top blight or other disease, and trunk cankers which greatly weaken the upper part of the tree. Trunk sprouting is usually more prolific during the rainy season at which time the cinchona trees undergo more active growth.

Conditions Favorable to Infection -- Susceptibility of different cinchona clones to top blight has been discussed previously. It might be added that the environmental, climatic, and cultural conditions prevailing in plantings have sufficiently great influence to alter the picture of apparent tolerance or vulnerability to infection by P. parasitica. Certain cinchona clones which are usually considered to be moderately tolerant to top blight have been observed to succumb to severe infections when wet weather persisted for an extended period of time and spore inoculum was heavy. Much of this inoculum was produced on infected Ledgers[4] and other susceptible cinchona clones in the vicinity.

At Finca El Naranjo the rainy season occurs from about May to November. Most of the approximately 175 to more than 200 inches of annual rainfall occurs during this period and nearly all cases of top blight occur at this time. During the months when rains are far less frequent and interrupted by suitable periods of intervening dry weather the incidence of this disease is very low.

Poor exposure of cinchona plantings favors top blight infections as a result of inadequate aeration and drying of trees. It has been noted consistently at Finca El Naranjo that the earliest and most severe outbreaks of top blight occur on western and northwestern slopes. During the rainy season daily morning sunshine lasts until about 11:00 a. m., followed by cloudiness and rains which usually persist throughout the afternoon, evening, and often at night. Adequate sunshine does not reach the western slope plantings until about 9:00 a. m., giving these plantings scarcely two hours of daily sunshine. This is inadequate for sufficient drying of the plantings to discourage infections, especially in dense plantings.

Humid conditions in plantings are also maintained by dense growth of trees and the presence of many tall weeds. Proper and timely thinning of trees from dense plantings and a reasonable amount of weeding would permit better air circulation through the planting, enhance drying, and help discourage many would-be P. parasitica infections.

Observation and Isolation of the Causal Fungus -- Under conditions of extended wet weather and heavy infection, the external signs of P. parasitica are easy to locate. Aerial sporangiophores arise in abundance from infection lesions and appear as white cottony tufts. Microscopic examination of freshly-collected top blight affected sprouts revealed that these natural infections produce abundant spore inoculum. Typical lemon-shaped sporangia were observed terminally on the aerial sporangiophores.

P. parasitica was readily isolated from leaf, twig, and stem specimens of sprouts and branches, either by the apple-isolation technique of Tucker (7) or by direct planting of surface-sterilized diseased tissue on agar medium. The fungus grew well on potato-dextrose agar, however, sporulation was rather sparse. Increased sporulation was induced by transferring the growth pellicle from the agar and floating it in a container of running water for 2 to 3 days. The harvested sporangia yielded zoospores when kept in water at reduced temperature. Typical top blight symptoms were produced in the laboratory on healthy cinchona sprouts sprayed with a

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[4]Term used synonymously with Cinchona ledgeriana and related clones.

spore suspension of P. parasitica isolated from natural infections.

Considering the large numbers of sporangia produced and zoospores released from natural top blight infections, and that the cycle from initial infection to production of sporangia can occur within about 20 hours, it is no wonder that severe top blight infections can occur in certain plantings within a few days if ideal conditions for infection exist.

Source of Inoculum -- A study was made to determine the source of inoculum responsible for the primary infections at the start of the rainy season and the infections which continue throughout this period. It was suspected at first that spores were produced from hold-over trunk girdle cankers, however, aerial sporangiophores were not observed to develop from these cankers even under prolonged wet conditions. It is not impossible that the cankers actually produce a limited number of sporangia under certain conditions.

A definite source of inoculum is the relatively few scattered light top blight infections found on sprouts during earlier periods of lesser rainfall. These infections yield a fair amount of spore inoculum at the start of the rainy season. The new spore crop splashed by rains to new infection courts continues a chain of many secondary infections.

The major source of inoculum accounting for heavy sprout infections during the early part of and throughout the rainy season is the soil. P. parasitica was isolated from a number of nursery and field soil samples by the apple-isolation method of Campbell (1). During heavy rains the fungus, presumably in vegetative form, is splashed with the soil onto cinchona trunks and sprouts growing at the base of trunks. The many resulting top blight infections produce more spore inoculum. This inoculum, together with continued splashing of soil-borne inoculum, account for numerous secondary top blight infections throughout the wet season.

Early in the wet season top blight infections are found almost exclusively on sprouts growing from the base of cinchona trunks. At this time few branch infections occur, presumably because inoculum is limited and because the soil-borne inoculum is not splashed so high by rains. Later, with considerable numbers of infected sprouts inevitably accompanied by production of much spore inoculum, the branches of susceptible trees do not escape infection.

TOP BLIGHT OF BRANCHES: Top blight of cinchona branches occurs under certain conditions and in severe cases can cause death of the tree. Many tree losses occur in plantings where wet weather persisting for several weeks encourages many rapid series of secondary branch infections. Severe top blight of branches presents a more dramatic picture than that of sprouts. Rapidly-spreading infections throughout the branch system during favorable weather may kill most of the branches on a tree within about ten days. Severe outbreaks of this type were noted in certain dense cinchona plantings where closely-growing trees and overlapping of branches formed a virtual moist chamber beneath. In many such cases, the conical apex of the tree, exposed to better aeration and drying, escaped severe infection whereas the branches below became completely infected and killed (Fig. 1, C). At Finca Los Ensayos in Costa Rica wet, cloudy, and foggy weather persists for many consecutive days uninterrupted by sunshine, and under these conditions even the apical branches of very susceptible trees are infected and killed as are the branches below. Most of the aerial part of the tree dies following death of the branches, however, many sprouts may develop from the base of the trunk and from the C. succirubra rootstock. The C. succirubra sprouts escape top blight infection owing to their tolerance to this disease, but the sprouts from Ledger and susceptible hybrid cinchona trunks soon become infected under the conditions described.

Unlike sprout infections, top blight of branches seldom progresses to the main trunk of the tree. Girdle canker of the main trunk in the upper part of the tree occurs far less frequently than at the base. One probable explanation for this is that the infections in the woody branch parts advance more slowly than they do in the more succulent terminal region and the branch is killed or rendered unfavorable to the pathogen before the fungus has had opportunity to reach the main trunk. Nectria sp. and other fungi often invade the weakened branch and probably limit further growth of P. parasitica.

Top blight of branches presents practically the same symptomatology as that of sprout infections. In both cases, although the more succulent terminal parts are more frequently infected, the fungus can infect the sprout or branch directly through the stem bark. Girdle of the stem in this region invariably results in wilt of distal parts of sprouts and branches.

GIRDLE CANKER

Trunk "girdle canker" of cinchona, caused by Phytophthora parasitica, accounts for heavy losses of susceptible trees. Susceptible varieties or hybrid clones are invariably the same as those susceptible to top blight. C. ledgeriana and related hybrid clones are most susceptible to the disease, whereas C. succirubra and related hybrid clones are very tolerant, although not

resistant. Natural cases of girdle canker were observed on trunks of C. succirubra and the causal fungus was isolated. The disease was produced on other C. succirubra trunks by inoculation.

That P. parasitica is responsible for both top blight and girdle canker was proved by isolations and by inoculations. The method employed to inoculate trunks was simple, rapid, and accountable for practically 100 percent infections on more than 1000 inoculated trunks of different clones. A plug of bark was removed with a No. 4 or 5 cork borer and a small amount of fungus culture, approximately 4 mm. by 4 mm., grown on potato-dextrose agar was inserted into the hole. The hole and culture were sealed over with a small dab of vaseline to maintain moisture and guard against desiccation of the culture before it had opportunity to initiate infection.

P. parasitica[5] culture No. NL123 isolated from a C. ledgeriana sprout infected with top blight produced typical girdle canker on inoculated trunks of C. ledgeriana, C. succirubra, and Hybrid Clone ZM. Conversely, typical top blight symptoms were produced on healthy C. ledgeriana sprouts in a moist chamber by spraying with spore suspensions of P. parasitica isolated from trunk girdle cankers.

Symptomatology -- Early infections of girdle canker are often difficult to detect because externally visible symptoms are generally lacking, especially if infection was initiated through uninjured bark. The color and texture of infected bark at this time are indistinguishable from that of normal bark. Sometimes reddish-brown droplets of exudate appear on the surface of infected bark. No sign of the causal fungus appears in this exudate which is probably cell fluid released from infected bark tissue following destruction of cell permeability. The fluid is subject to rapid oxidation and changes to the reddish-brown color on contact with air. When the exudate dries it imparts a bronze coloration to the bark surface.

New girdle canker infections are more readily located at the base of top blight infected sprouts after the infection has progressed to the trunk. Removal of the bark surface with a knife reveals the brown infected bark tissue beneath. The advancing regions of the canker show evidence that active infection and destruction of healthy bark tissue is in progress. These regions may be uniformly light brown or may present a muriform effect made up of variable shades of brown and tan. Irregularly sized and shaped cream-colored or tan patches of bark are surrounded by a more or less definite perimeter of dark brown tissue where fungus growth was apparently more active and the bark killed more quickly. Eventually, as the bark and probably the fungus are killed, the bark becomes uniformly dark brown. Attempts to isolate P. parasitica from the older portion of the canker were not successful.

Advance of infection is not uniform around the perimeter of the canker. Fungus growth may cease entirely at certain edges of the canker, the bark tissue may be killed, and new callous tissue may develop normally. The fungus remains very active in other parts of the canker and advancing infections may radiate from various regions of the canker circumference, often resulting in cankers of very irregular shapes. Advance of canker infections is usually most rapid upward and somewhat slower downward. Rapidity and extent of canker development depends on degree of tree susceptibility and on the strain and viability of the fungus. For instance, canker infections on artificially inoculated trunks of very susceptible Hybrid Clone Z-56 developed nearly 50 cm. above and below the site of inoculation within 3 months; during the same period the cankers on inoculated trunks of very tolerant Hybrid Clone Z-630 extended scarcely 2.5 cm. above and below the site of inoculation.

Lateral advance of the canker is usually slower than vertical progress, but at times may exceed vertical growth. The two lateral edges of the canker advance around the trunk circumference and unite to form a complete trunk girdle. Inoculated trunks of Hybrid Clone Z-56, averaging at least 20 cm. in circumference at the site of inoculation, were completely girdled by girdle canker within one month of inoculation; on inoculated trunks of Hybrid Clone Z-630 the lateral extent of the canker averaged less than 2 cm. in three months.

Complete trunk girdle accompanied by disruption of normal conducting and translocating activity between roots and branches causes rapid decline of the upper portion of the tree. This decline is manifested within several weeks (sooner during the dry season) of trunk girdle as discoloration of branch foliage especially at the apex of the tree. These symptoms of apparent tree starvation appear as variably-colored leaves ranging from chlorotic to orange and deep red. Newly-emerging foliage is abnormally small and soon subject to the same discoloration and to wilting. Gradual general wilting of young and old leaves occurs until the entire foliage

[5]Identification confirmed by C. M. Tucker.

system is wilted and the branches and trunk die. The bark on dead cinchona trunks is tightly united with the wood and is extremely difficult to harvest.

Although the aerial part of the tree is killed, the C. succirubra rootstock usually remains alive and produces new sprouts. Girdle canker infections from grafted trunks seldom extend more than one inch into the C. succirubra part; however, a number of trunk cankers, apparently caused by a more potent fungus strain, were observed to continue well below the graft union (Fig. 2, C). Some girdle cankers extended at least 8 inches into the succirubra region. Since these cankers did not yet reach the soil, the possibility of the infections having occurred via infected roots was discounted.

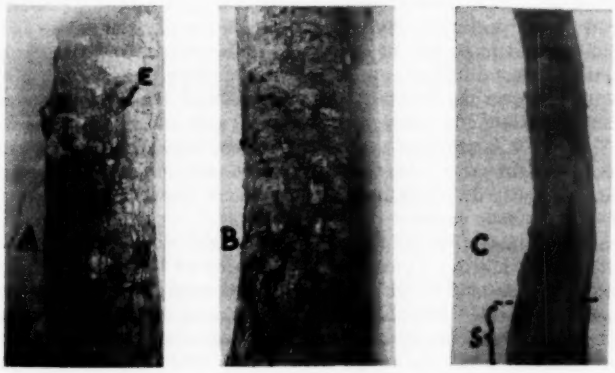

FIGURE 2. Cinchona ledgeriana trunks with "girdle canker" symptoms. A. Relatively early canker showing droplets of exudate (E). B. Advanced canker showing shrunken nature of bark. C. Surface of bark removed to show canker extending into the C. succirubra base (S) of the trunk.

Advanced girdle cankers can often be detected before the tree shows foliage symptoms of decline by examination of trunks. These cankers are usually discolored by a conspicuous reddish brown exudate. At or near the active advancing edges of the canker the exudate is usually in the form of droplets (Fig. 2, A). The older part of the canker is usually covered with a film of exudate. This, when dried, gives the cankered bark a noticeable rust to bronze color.

Independent of release of exudate the bark of older cankers becomes darker than normal bark. This occurs when the bark tissue is killed and changes in color from nearly white to dark brown. Death and drying of the cankered tissue causes the bark to become noticeably shrunken (Fig. 2, B). A definite margin is usually observable at the inactive edges of the canker, where the bark is shrunken and new bark callous tissue from the healthy area begins to ward in the canker. Such a margin between cankered and healthy bark is usually not sharply defined in the region where the canker is actively advancing.

Trunk girdle canker infections penetrate cinchona bark and enter but a short distance into the sapwood. The discoloration which accompanies infection seldom penetrates more than about 2 millimeters into the sapwood. In the older portion of the canker the wood is dead and discolored more deeply but not as a result of the pathogen directly. P. parasitica was not isolated from the older portion of the canker, but was isolated from the brown discolored sapwood at the advancing edge of the canker.

Conditions Favorable to Girdle Canker -- The same conditions which favor top blight infections also favor girdle canker infections. This trunk disease occurs far more frequently and with more serious losses during the wet, humid season. Infections are far more common in dense, poorly-aerated plantings on poorly exposed sites than in more open stands. Planting distances between trees and the amount of subsequent thinning determine the density of trees in plantings.

Trunk infections occur in greater numbers in plantings where the bases of trees are prevented from adequate drying. Presence at the base of trunks of pruned branches and lush growth of weeds helps maintain wet, humid conditions in the trunk environment for a sufficient period of time to permit initiation of infection. These conditions also help maintain spore viability for a longer time.

Growth of small weeds and a shallow soil covering of old leaves are desirable as long as they are kept low and do not surround the trunk closely. Very clean weeding or other practice which

leaves the soil exposed near the trees should be avoided. Heavy rains splash the soil onto cinchona trunks and sprouts and an excellent opportunity is provided for the soil-borne pathogen to infect the trees. Soil covered by leaf residue or small weed growth has less chance of being splashed by rains.

Manner of Natural Trunk Infection -- There are two main avenues of entry for the fungus causing girdle canker, namely, directly through the injured or non-injured bark, and within stems of top blight infected sprouts. Highly susceptible cinchona clones are readily prone to both manners of infection. The more tolerant clones are more prone to infection via the top blight infected sprouts than via the non-wounded bark.

An inoculation study was conducted to determine the relative ease of infection via both the injured and non-injured bark. In this study inoculations were performed as follows: Sterile corn kernels partly submerged in potato-dextrose agar became infected by P. parasitica planted on the agar. An infected kernel with adhering agar culture was placed on a 2 by 2-inch square of wetted cotton and appressed to the trunk with the kernel and inoculum adjacent to the bark. The inoculum and cotton were kept in place with wrappings of strips cut from a large tropical leaf and string. Ten trunks of Hybrid Clone ZM and five trunks of C. ledgeriana were inoculated at the site of a bark wound made with a knife. The same number of each were inoculated by appressing the inoculum directly on a region of healthy bark showing no evidence of injury. Inoculations were checked 20 days later.

All wounded and non-wounded trunks of C. ledgeriana developed girdle cankers extending 8 to 10 cm. from the site of inoculation within 20 days. Some of these trunks were 60 percent girdled. All the wounded trunks of Hybrid Clone ZM developed girdle canker approximately half the size of those on Ledger trunks. The ZM trunks inoculated over non-wounded bark developed principally local shallow infections despite the abundant inoculum in constant contact with the bark for 20 days under conditions favorable for infection. This is a fairly severe test which is usually not duplicated under natural conditions. This slow take of infections on Hybrid Clone ZM shows how much more difficult it is for natural infections to occur through non-injured bark of tolerant clones than of more susceptible cinchona trees.

Although many natural girdle canker infections occur via top blight infected sprouts, all sprout infections do not reach the trunk. Infection of large woody sprouts is often limited to the terminal region and does not advance to the trunk. Small, succulent sprouts are much more dangerous to the trunk than the large, woody sprouts because they are far more prone to top blight infections and these infections continue to the trunk more frequently.

Many cases of girdle canker were observed on cinchona trunks free of sprouts. Nearly all of these trunks became infected within two feet of the soil. The more susceptible trees appear to have become infected through the uninjured bark. Presence of mechanical wounds or other bark openings increases considerably the chance of trunk infection. During the heavy rainy season, elliptical bark eruptions averaging 5 to 8 mm. long are very numerous at the base of many cinchona trunks. They occur both on C. succirubra rootstock and on grafted trunks. These bark eruptions, which apparently are enlarged lenticels, serve as excellent portals of entry for the pathogen and occur in the region of the trunk most liable to soil splashing. Many new cases of girdle canker during the wet season were traced directly to natural bark eruptions.

Source of Inoculum: The sources of inoculum for girdle canker infections of cinchona trunks are about the same as described for top blight infection of sprouts. The major source of inoculum at the start of and during the rainy season is the P. parasitica contaminated soil splashed onto trunks during rains. The presence of the pathogen in nursery and field soils was proved by isolation and its pathogenicity proved by trunk inoculation.

Strain Variations of the Pathogen -- Field observations and inoculation studies showed that strains of P. parasitica exist. Isolates of the pathogen from the soil, from top blight infected sprouts and branches, and from trunk girdle cankers appear identical in culture, but the growth rate of certain isolates differs significantly in culture. Isolates also differ with respect to the rate of growth and size of cankers produced on inoculated cinchona trunks. For instance, P. parasitica[5] Culture NL153 secured from the soil by the apple-isolation technique (1) (7) proved to be a very potent strain. It grew more rapidly on agar medium than did most other isolates and produced larger cankers on inoculated trunks than did practically all other isolates.

C. succirubra is seldom infected by P. parasitica under natural conditions. Various P. parasitica isolates inoculated into C. succirubra trunks produced infections which advanced only several centimeters from the site of inoculation and were walled in by new callous tissue. One group of C. succirubra was observed in which a number of trees were infected with girdle cankers. The canker on one trunk was measured and found to extend 70 cm. along the long axis of the trunk which was nearly girdled. P. parasitica isolate NL444 was cultured from the advancing edges of this canker. This isolate and soil isolate NL153 were inoculated into trunks of C.

C. ledgeriana, C. succirubra, and Hybrid Clone ZM. Both produced cankers of about the same size on respective trees, thus exhibiting similar strain potency which was greater than that of other cultures of P. parasitica isolated from various sources.

Certain cinchona clones may differ in their response to infection by different strains of the pathogen. Ledger Hybrid Clone H-1229 appeared to be moderately tolerant to P. parasitica isolate NL153 inoculated into five trunks. The girdle cankers produced on these trunks were much smaller than those produced by the same isolate on all other Ledger clones during the same interval of time. During the inoculation study, Clone H-1229 became infected with natural girdle canker on all five trunks and in a short time these cankers outgrew the cankers produced by isolate NL153. The fungus causing the natural infections was undoubtedly a more potent strain than isolate NL153, at least to Clone H-1229. Similar differences between natural infections and infections resulting from inoculation were observed on two other hybrid clones, Z-92 and N-3.

Influence of Girdle Canker on Quinine Production -- Assays of diseased and healthy cinchona bark samples were made to determine whether girdle canker has any direct influence on quinine production. Bark samples were collected from five-year old trees of Hybrid Clone ZM growing in the same planting. Bark was harvested from the entire trunk of 10 healthy trees and from 10 trees with natural girdle cankers involving approximately one-half the trunk circumference. It was estimated that the average infection was seven or eight weeks old. After drying, grinding, and uniformly mixing all the bark of each lot, representative samples were submitted for complete assay to the Department of Chemistry at the Instituto Agropecuario Nacional[6] in Guatemala City.

The bark samples from healthy and from girdle canker infected trunks assayed approximately the same, in fact, the sample from diseased trees assayed slightly higher than that of healthy trees. This indicates that girdle canker of relatively short duration has no deleterious effect on the quinine producing capacity of the bark. It is assumed that in the case of slowly-progressing infections, as found on trunks of more tolerant cinchona clones, the bark of a diseased tree may show a significant decrease in quinine content. This would probably not be due to any direct antagonistic effect of the fungus on the alkaloid-synthesizing activity of he bark, but more likely is the result of gradual reduction in tree vigor over a period of months.

SUMMARY

Phytophthora parasitica Dast. causes two dissimilar phases of disease to cinchona trees in field plantings. One, called "top blight" is a disease of sprouts and branches; the other, called "girdle canker", is a trunk disease.

Spread of the disease is favored by cool, wet, humid weather, dense plantings, poor exposure, and other conditions which prevent adequate drying of plantings.

There is marked difference in susceptibility to the pathogen among different species, varieties, and hybrid clones of cinchona. Cinchona ledgeriana and related hybrid clones show high susceptibility; C. succirubra and related hybrid clones show considerable tolerance to most strains of the pathogen.

Girdle canker develops both from top blight infected sprouts and through direct infection of the injured and non-injured bark. Bark injuries, lenticels, and other bark openings predispose the trunks to easier infection.

P. parasitica is soil-borne, which accounts for many top blight and girdle canker infections resulting from splashing of contaminated soil onto sprouts and trunks by rains.

Strain variations of the pathogen exist, and significant differences in pathogenicity were observed among strain isolates.

Trunk girdle canker of short duration apparently has little, if any, adverse effect on the production of quinine alkaloids in cinchona bark.

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PHYTOPHTHORA CINNAMOMI ON AVOCADO IN HONDURAS

George A. Zentmyer and Wilson Popenoe

Phytophthora cinnamomi Rands was recently isolated from feeder roots from diseased avocado trees in Tegucigalpa, Honduras. The roots were shipped under quarantine permit to Riverside, California, in September, 1950.

The roots were taken from two Atlixco hybrid trees growing on West Indian rootstock, in an avocado nursery at the Escuela Agricola Panamericana in Tegucigalpa. These trees were showing severe disease symptoms, similar to those of avocado root rot occurring in Peru (1), Puerto Rico (2), and California (3, 4). The soil contains considerable clay and is very difficult to drain; many of the nursery trees, as well as all trees in a five-acre planting on similar soil, have died in the past few years. The majority of the trees succumb to the disease between two and five years of age. The trees from which P. cinnamomi was isolated were grown from seed on the property, indicating that the fungus is native in the soils of that region.

The roots were packed in sphagnum and wrapped in Goodyear Vinyl film; they arrived in California in good condition after being in transit nearly two weeks. The fungus was isolated by the usual method employed in this laboratory, which involves washing entire 1 cm. long pieces of small roots in water, washing briefly in 70 percent alcohol, blotting, and planting on cornmeal agar. P. cinnamomi appeared on 6 of 30 root pieces cultured.

This is apparently the first record of this fungus in Honduras.

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VERTICAL DISTRIBUTION OF PHYTOPHTHORA CINNAMOMI IN THE SOIL UNDER LITTLELEAF-DISEASED SHORTLEAF PINE

W. A. Campbell

In a study conducted from December 1948 through February 1949, Phytophthora cinnamomi Rands was isolated from 41.7 percent of the soil samples taken under littleleaf-diseased shortleaf pine (Pinus echinata Mill.), from 14.6 percent of the samples taken under healthy trees on littleleaf sites, and from only 4.6 percent of the samples taken under healthy trees on littleleaf-free sites. [1] Soil samples were taken to a depth of six inches and assayed for the presence of P. cinnamomi by placing soil to be tested in holes in apples. [2] In order to provide information on the vertical distribution of P. cinnamomi in the soil under littleleaf trees, 31 samplings were made to the depth of 12 inches under 28 trees in the following manner: A pit approximately 18 inches wide, and 18 inches deep with one face vertical was dug under each tree to be sampled. By means of a foot ruler toothpicks were stuck in the vertical soil face, one inch apart, starting one inch below the soil surface and continuing to a depth of 12 inches. Two soil samples were taken with a half inch hollow tube to the right of each toothpick. Each of these samples was placed in a hole in an apple. Likewise two samples were taken to the left and placed in holes in a second apple. One apple was marked A, the other B. Verification of the presence of P. cinnamomi was made by plating bits of rotted apple tissue on cornmeal agar.

Soil samples were taken during February and March 1949 under trees with typical or advanced littleleaf in the general vicinity of Athens, Georgia.

Phytophthora cinnamomi was isolated 1 to 9 times from 26 of the 31 pits. The highest number of recoveries came from those pits located in known P. cinnamomi-infested spots as determined by a previous study. However, it was not possible to dig all the pits in areas where the fungus was concentrated, and consequently no P. cinnamomi was isolated at any depth from five of the pits. The fungus was isolated 98 times from the samples taken at various depths, or 26 percent of the 372 samples taken at depths ranging from 1 to 12 inches. Although 4 individual soil samples were taken at each depth and placed in holes in 2 apples, positive results from one apple was evidence of the presence of the fungus at that depth.

FIG. 1 - FREQUENCY OF ISOLATION OF PHYTOPHTHORA CINNAMOMI FROM SOILS UNDER LITTLELEAF-DISEASED SHORTLEAF PINE AT DEPTHS FROM 1 TO 12 INCHES

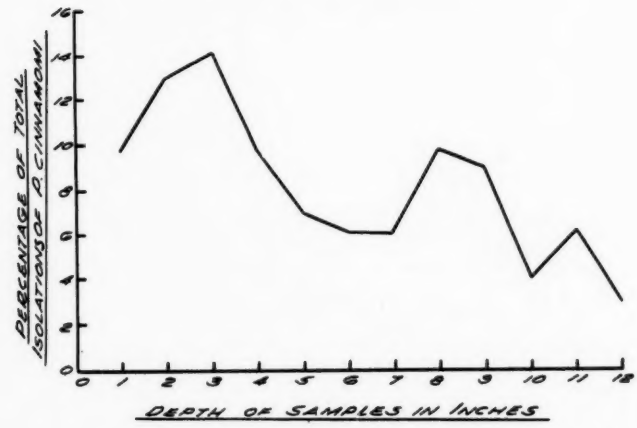

FIGURE 1

[1] Campbell, W. A. Relative abundance of Phytophthora cinnamomi in the root zones of healthy and littleleaf-diseased shortleaf pine. Phytopath. 39: 752-753. 1949.

[2] Campbell, W. A. A method of isolating Phytophthora cinnamomi directly from soil. Plant Dis. Reptr. 33: 134-135. 1949.

The frequency of isolation of Phytophthora cinnamomi expressed in percentage of the total number of positive isolations of the fungus at different depths is given in Figure 1. The fungus was most abundant at the 2- and 3-inch depths, falling rapidly to a low point at 6 and 7 inches, after which it was again more abundant at 8 and 9 inches. Even at the depth of 12 inches the fungus occurred to a limited extent.

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OAK WILT FUNGUS PATHOGENIC TO CHINESE CHESTNUT [1]

T. W. Bretz

The isolation of Chalara quercina, the fungus that causes oak wilt, from Castanea mollissima was reported recently in the Plant Disease Reporter. [2] Inoculations with an isolate of the fungus from Chinese chestnut, as well as with an isolate obtained from northern red oak (Quercus boreal is), into a limited number of C. mollissima trees, have demonstrated the pathogenicity of the fungus to this species.

Four trees of C. mollissima, accession FP-1227, were inoculated with the isolate from chestnut. One tree of the same accession, and one tree of accession FP-1161, were inoculated with the isolate from northern red oak. Mycelium and spores from agar slants were inserted into cuts penetrating the outer rings of wood of the main stem. The incisions were covered with moist cotton and wrapped in aluminum foil to prevent too rapid drying out. The trees were two to three feet in height and from one-half to three-quarters inch caliper, growing out of doors in nursery rows. All the trees inoculated with each isolate were showing evidence of wilt infection 27 days after inoculation. Symptoms included wilting and curling of leaves, bronzing of foliage, and defoliation in the more advanced stages. There was no apparent sapwood discoloration.

Twig samples from all trees were cultured and Chalara quercina was isolated from three of the four trees inoculated with the chestnut isolate and from both trees inoculated with the isolate from northern red oak. The reisolates appeared to be identical with the isolates used for inoculation.

Although the number of trees involved in this preliminary work is small, the results indicate that the oak wilt fungus is a virulent pathogen in the C. mollissima accessions tested and that interspecies transfer of the fungus from oak to chestnut may occur.

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[1] Contribution from the Department of Botany, Missouri Agricultural Experiment Station, Journal Series No. 1236.

[2] Bretz, T. W. and W. G. Long. Oak wilt fungus isolated from Chinese chestnut. Plant Dis. Reptr. 34: 291. 1950.

DIE-BACK OF SYCAMORE

G. E. Thompson

A die-back of sycamore (Platanus occidentalis L.) has been observed in the vicinity of Athens, Georgia, during the past two summers. The area of heaviest infection was along a small stream, where about 40 percent of the trees were in various stages of degeneration, from those with one or two dying branches to dead trees.

The disease appeared to have started in the small branches and progressed back to the large branches and finally into the trunk. Wilted and dead leaves were present on many branches in July when the observations were made. A number of the affected trees had dead tops with a few basal living branches (Fig. 1). Internally the woody tissues of the branches and trunks showed brown streaks of varying lengths. In cross section these were in the form of irregular discolored areas which were not confined to any particular part of the stem (Fig. 2). Stained microtome sections of the affected wood showed long, broad hyphae in the vessels.

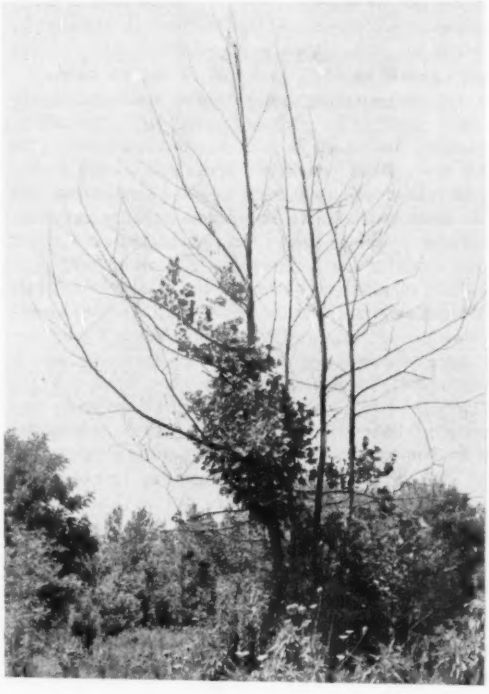

FIGURE 1. Typical late stage of sycamore die-back.

FIGURE 2. Cross-section of 4 inch stem showing discolored areas.

Pieces of recently killed branches, some bearing withered leaves, were placed in moist chambers. In about two weeks pycnidia with conidia were produced abundantly in the bark tissues and also in the leaf petioles. A fungus was isolated readily in culture from the discolored wood and leaf petioles. Pycnidia with conidia similar to those found on the diseased twigs were produced in the cultures. The best development of these took place on and around the pieces of wood used for making isolations. The fungus has been identified as Diplodia natalensis Evans.

Inoculations were made on June 21, 1950, by inserting bits of mycelium and conidia under the bark on small healthy trees. After placing the inoculum in the wound, the bark was bound back into place with cellophane tape. Twelve inoculations were made on branches which varied in diameter at the point of inoculation from one-fourth to three-quarters inches, and two were made on the stems of small trees 1 1/2 and 2 1/2 inches in diameter. Five controls were wounded similarly but the inoculum was omitted.

On July 3, 1950, all inoculations showed infection. On the smaller branches with thin bark, these appeared as dark, elliptical, slightly sunken lesions from 1 to 3 inches in length. On the larger branches and stems with thicker bark, the discoloration was scarcely noticeable. Internally, dark streaks were present in the xylem and extended above and below the point of inoculation for distances of 2 to 16 inches. The fungus was reisolated without difficulty. The controls showed some browning of the bark and underlying xylem, but it was limited to the immediate vicinity of the wounds.

Siggers (1) has reported a disease on sycamore in Louisiana with somewhat similar symptoms but made no suggestion as to its cause.

It is quite probable that anthracnose, which has been rather severe on sycamores at Athens, has decreased the vitality of the trees, so that D. natalensis has been able to make rapid progress on the weakened trees.

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BANANA MOSAIC IN BORNEO AND ITS RELATIONSHIP TO ABACÁ MOSAIC IN THE PHILIPPINES

Otto A. Reinking [1]

On October 11, 1950 a small planting of banana varieties was inspected for disease on the Table Estate of the Borneo Abacá Ltd. at Tawau, British North Borneo. Typical banana mosaic was observed on plants of the Lady Finger and Cavendish varieties. The disease on the Lady Finger variety was characterized by a distinct yellow and green mottling of the central younger group of leaves. The characteristic leaf streaking of yellow in with the green of the leaf as described by Magee (2) for infectious chlorosis on bananas in Australia was present. Badly affected leaves, in addition to the streaking, had dried browned edges along with a shrivelling and splitting of the leaf blade. A green, streaked mottling along with brownish black streaks was present on the affected leaf petiole. Affected plants were stunted. The disease resembles closely descriptions given by Wardlaw and McGuire (8) for the so-called Brazilian disease on Cavendish and Giant Fig banana varieties in Brazil. Similar troubles were reported by Wardlaw in Trinidad B. W. I. (7), Ogilvie in Bermuda (3), and Jensen in Hawaii (1). The mosaic type of trouble described in this paper appeared to be different from those described in August 1949 (5).

The writer in August, 1949 in Borneo (5) made a report of banana mosaic and possibly other virus troubles, which observations indicated were apparently different from the mosaic of abacá in Davao, Philippine Islands. The fact, however, that these troubles on bananas apparently are of a virus nature and that at least one closely resembles some symptoms of bunchy-top in abacá shows the need of further study. In addition to this possible connection, the mosaic described in this paper resembles somewhat mosaic of abacá in the Philippines. Until virus transmission studies are made of all suspected virus troubles reported on bananas in Borneo (5) their true identitity and relationship to abacá virus troubles cannot be stated.

The mosaic of the banana varieties studied in October, 1950 on the Table Estate at Tawau, British North Borneo had symptoms in part similar to, but apparently different from those of abacá mosaic in the Davao area on the Island of Mindanao in the Philippine Islands (4). Virus-infected Commelina nudiflora and Paspalum conjugatum, along with an aphid closely resembling Aphis gossypii, are commonly found in most abacá plantings in Borneo. This aphid and Aphis maydis have been shown capable of transmitting a virus of Commelina to celery, pepper, banana, and corn by Wellman in Florida (9). Magee, (2) using Aphis gossypii as a vector, transmitted infectious chlorosis from the Cavendish banana to Musa ensete and to a seeded Musa sp. From the latter species the virus was transmitted by the same aphis to the Cavendish and Gros Michel varieties of banana, to abacá, to canna (C. indica) to cucumber, to squash, and to tomato. Magee, (2) considered the virus to be a strain of Cucumis virus 1. Silberschmidt and Nobrega (6) in Brazil showed that a virus from Commelina and from banana could be transmitted to Solanaceous plants. What relationship the Commelina or Paspalum virus-affected plants had to the mosaic on banana in the Borneo planting of course could not be stated. Transmission studies would have to be made to determine this point.

During the two inspection trips made in 1949 and 1950 the writer never observed mosaic of abacá in Borneo. In the Davao Area of the Philippines, where abacá mosaic is rampant, Paspalum conjugatum virus-infected plants are suspected of carrying the abacá mosaic virus. This as yet has not been definitely proved. In Borneo Paspalum conjugatum virus-infected plants are present in abacá plantations, as apparently is Aphis gossypii, one of the vectors of abacá mosaic, but as yet mosaic of abacá has not been observed.

A mosaic type of virus trouble on banana plants is present in Borneo. Because of this fact and the presence of a virus-infected weed suspected of carrying abacá mosiac in the Philippines and the presence of mosaic virus aphis carriers a careful check should be maintained for detection of abacá mosaic appearance in Borneo. The entire banana virus complex and the virus complex on weeds in Borneo should be investigated and their relation to disease in banana and abacá established (5).

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U. S. DEPARTMENT OF AGRICULTURE, OFFICE OF FOREIGN AGRICULTURAL RELATIONS

BUCKSKIN FRUIT SYMPTOMS ON SWEET CHERRY TREES ON MAHALEB STOCK

Carl W. Nichols and George Nyland

It has been reported by Rawlins and Parker (1) that in Green Valley, Solano County, California, sweet cherries on Prunus mahaleb stock showed resistance in the field to buckskin disease or escaped natural infection. But if even a single sweet cherry root was produced above the P. mahaleb union, this was sufficient to confer susceptibility upon a tree. When diseased scions were placed on healthy sweet cherry trees on P. mahaleb stock, however, symptoms were produced. The predominant symptoms were severe chlorosis and marked weakening or death of the affected trees.

When sweet cherry trees on Mazzard stock become infected with the buckskin virus, they produce characteristic fruit symptoms but not markedly chlorotic foliage. These fruit symptoms have not been reported as occurring on trees on P. mahaleb stock as far as is known.

During the 1950 growing season, we observed that ten sweet cherry trees of the Bing variety on P. mahaleb stock on one property in Green Valley produced fruits that were indistinguishable from those typically produced by trees on Mazzard stock when infected with the buckskin virus. This condition was also reported as occurring on two other properties in the same district but these were not seen by the writers. As the season advanced the limbs which produced buckskin fruits began to look unthrifty and the leaves became chlorotic. Where the sweet cherry scions had been placed high on P. mahaleb, in the recommended manner, some were dead by the middle of September and others were dying as they do when artificially inoculated with diseased scions. All affected trees were examined and no roots were found above the graft union. This union, in some cases, was as much as three feet above the ground level.

Whether the decrease of field resistance in Green Valley to buckskin of sweet cherry on P. mahaleb stock is only temporary is not known as yet. No satisfactory explanation of the above condition is available at this time.

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YELLOW LEAF ROLL OF PEACH

George Nyland and Archie Schlocker

The Agricultural Commissioner's Office and the University of California Extension Service, both of Yuba County, independently called our attention last spring to a disease of clingstone peaches which we are tentatively calling Yellow Leaf Roll. There appears to be no varietal resistance to the disease. In the affected area the disease has been observed in the field on at least seven of the 12 common varieties of clingstone peaches, and produces practically identical symptoms on each.

Leaf symptoms consist of marked yellowing, rolling of leaves upward and inward, some marginal scorching and some marginal and laminal necrotic spotting resulting in ragged leaves, a tendency for the leaves to curve back toward the stem, and, later in the season, marked vein swelling. Affected leaves drop earlier than healthy leaves.

There is a failure of fruit production or an arresting of fruit development on affected branches. The entire tree or only part of it may be involved. The disease seems to advance upward from the base of the main scaffold limbs. Frequently terminal, healthy-appearing flags may be present on an otherwise completely diseased limb. The contrast between the healthy and diseased portions of the tree is very striking.

According to information supplied by several growers in the affected area, the disease has spread rapidly during the last three years. It was reported that in one block of about 900 trees 8 diseased trees were found in 1948, 25 additional trees in 1949, and 80 additional trees in 1950. In another orchard of about 20 acres 3 diseased trees were observed in 1948, 17 diseased trees were removed in 1949, and in 1950, an additional 39 trees were found. This serves to illustrate the seriousness of the trouble.

Surveys by State and county inspectors have shown the disease to be concentrated in about 20 orchards in a small area in Yuba County and occurring to a lesser extent in 10 orchards in adjoining Sutter County.

From the first observations made by us last spring, it was strongly suspected that yellow leaf roll was a virus disease. The symptom picture does not fit any known virus or other trouble on peach. It resembles in some respects buckskin or western X-disease of peaches which also occurs in the same area. However, in their typical forms the two diseases can be distinguished readily from each other. Intermediate symptom types have been observed and final identification of these must await further investigation.

On July 6, 1950, current season buds and older spurs taken from shoots showing leaf symptoms were budded to one-year old trees in the field. Varieties inoculated were Gaume, a clingstone, and Elberta and Rio Oso Gem, freestones. Additional buds were inserted on July 22. Indications of transmission were obtained October 19, 1950. A low shoot on one tree of Gaume and two basal suckers of one tree of Elberta showed typical symptoms. Several other inoculated trees of all three varieties were strongly suspected at the time this was written. There seems little doubt that the causal agent is a virus.

UNIVERSITY OF CALIFORNIA, AGRICULTURAL EXPERIMENT STATION, DAVIS, AND BUREAU OF PLANT PATHOLOGY, CALIFORNIA STATE DEPARTMENT OF AGRICULTURE, SACRAMENTO

DISEASES OBSERVED ON BRAMBLE FRUITS IN THE PACIFIC NORTHWEST

Edward K. Vaughan[1], Folke Johnson[2],
R. E. Fitzpatrick[3], and Richard Stace-Smith[4]

A survey of cane fruit diseases was conducted in Oregon, Washington, and British Columbia, west of the Cascade Mountains, during the period of June 5 to 13, 1950. The primary purpose of the survey was to determine what virus diseases were present in the area, where and on what varieties they were most prevalent, and the relative severity of each. Observations were also made for diseases of pathogenic and physiologic origin.

VIRUS DISEASES

Three diseases of definitely virus origin were observed.

MOSAIC (Marmor rubi Holmes)[5], was present, either alone or in conjunction with ring spot, throughout the area covered by the survey. In some cases the disease was not causing apparent devitalization or stunting of the plants or reduction in yield. Severity of infection, varieties on which the disease was observed, and locations were as follows:

(a) Symptoms severe, accompanied by dwarfing of the plants:

Black raspberry	Unknown variety	Gresham, Oregon
Boysenberry		Pitt Meadows, British Columbia

(b) Symptoms moderate, not accompanied by dwarfing:

Red raspberry	Washington variety	General throughout the Northwest
	Cuthbert	General throughout the Northwest
	Latham	Pitt Meadows, Lulu Island, British Columbia
	Sunrise	Mount Vernon, Washington
	Wn 318	Mount Vernon
	Wn 327	Mount Vernon
	Taylor	Mount Vernon; and Mission, British Columbia
	Newbergh	Langley, British Columbia
Black raspberry	New Munger variety	Vancouver, British Columbia

LEAF CURL (Corium rubi Holmes), because it produces a marked dwarfing, malformation and reduction of productivity, is usually eliminated from the field as quickly as its presence is discovered by the grower. It is, therefore, seldom seen in the field. Curl (probably var. alpha H) was observed on red raspberries only, on the variety Taylor at Vancouver, Washington and on the variety Tahoma at Puyallup, Washington.

DWARF (Nanus loganobacci Holmes) was seen in a single field of Loganberry at Albany, Oregon.

OTHER DISEASES APPARENTLY OF VIRUS NATURE:

Three other troubles were observed which appeared to be of virus nature and will be included under the general heading of virus diseases.

RING SPOT, observed only in red raspberry, is characterized by roughly circular areas of light green with centers of nearly normal dark green. The rings vary from 3 mm to 15 mm in diameter, being usually larger and less numerous on the newest leaves, and smaller but more numerous on the older leaves (Figures 1 and 2). The disease is frequently associated with a mosaic which, however, is apparently not yellow blotch curl (Can. Jour. Res. C 16: 118-124. 1938), a disease which also produces ring spot symptoms. It is not unusual to observe ring spot on the newest leaves and mosaic on the older leaves of the same cane. No apparent stunting or

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[5]Virus nomenclature from Holmes, F. O., The Filterable Viruses, Supp. #2, Bergey's Manual of Determinative Bacteriology 1948 - Williams and Wilkins Co., Baltimore.

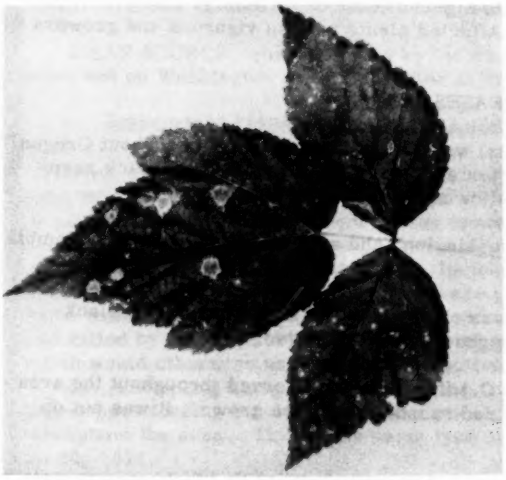

FIGURE 1. Ringspot of red raspberry: Early stage (Washington variety).

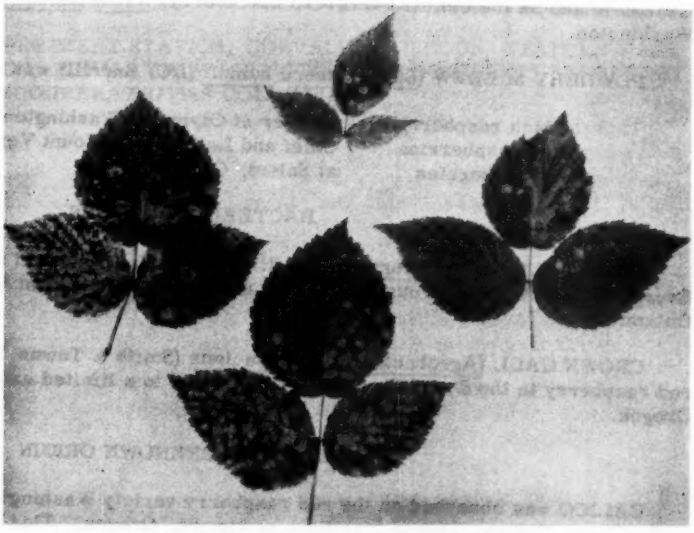

FIGURE 2. Ringspot of red raspberry: Later stage (Washington variety).

loss of vigor is associated with the disease. Varieties on which the disease was observed, and their location, were as follows:

Variety Washington	General throughout Oregon and Washington
Tahoma	Puyallup, Washington
Chief	Mount Vernon, Washington
Wn #324	Mount Vernon, Washington
Willamette	Oregon City, Oregon

PURPLE STUNT, a disorder entirely distinct from blackberry dwarf, was observed on Thornless Loganberry at Woodburn, Oregon. Severely affected plants are dwarfed, the longest canes reaching a maximum of 4 to 6 inches, and remain purplish in color throughout the season. Less severely affected plants are stunted and unthrifty. There is no rosetting, in contrast with the marked rosetting that characterizes dwarf. According to the owner of the field, severely affected plants die during the next winter.

YELLOWS was observed on the Munger variety of black raspberries in the vicinity of

Lacomb, Oregon. The only symptom is a light yellow-green color of the foliage which contrasts sharply with the dark green of the normal plants. Affected plants remain vigorous and growers report they retain full productivity.

FUNGUS DISEASES

ANTHRACNOSE (Elsinoë veneta (Speg.) Jenkins) was observed commonly throughout Oregon and Washington, particularly where black raspberries are grown. All varieties of black raspberries are susceptible. It was also seen on the following varieties of red raspberry:

Washington: Throughout Oregon and Washington, and at Huntington, British Columbia
Lloyd George: Huntington

LEAF AND CANE SPOT (Septoria rubi West) was observed in all fields of trailing blackberries (including Logan, Boysen, and Young) throughout the area visited.

YELLOW RUST (Phragmidium rubi-idaeae (D.C.) Karst.) was observed throughout the area wherever the Washington and Cuthbert varieties of red raspberries were grown. It was not observed on any other varieties.

CANE AND LEAF RUST (Kuehneola uredinis (Link) Arthur) was found on blackberry variety Chehalem and on the cutleaf evergreen blackberry, Rubus laciniatus, throughout Oregon and Washington.

POWDERY MILDEW (Sphaerotheca humuli (DC) Burrill) was seen on the following varieties:

Black raspberries	Munger at Olympia, Washington
Red raspberries	Chief and Latham at Mount Vernon, Washington
Boysenberries	at Salem, Oregon

BACTERIAL DISEASES

CANE GALL (Agrobacterium rubi Hildebrand) was observed on Boysenberry, Loganberry and Evergreen Blackberry throughout Oregon and Washington and on Evergreen Blackberry in British Columbia.

CROWN GALL (Agrobacterium tumefaciens (Smith & Towns.) Conn) was found on Cuthbert red raspberry in the Gresham area of Oregon and to a limited extent on Loganberry at Woodburn, Oregon.

DISEASES OF UNKNOWN ORIGIN

CALICO was observed on the red raspberry variety Washington at Salem, Oregon, on Cuthbert at Gresham, Oregon, and on Marlboro at Aurora, Oregon. This trouble is characterized by a lack of green pigment along the veins of the leaves of the current season's growth. When only the lower leaves are affected the trouble may be entirely due to faulty nutrition. There is another type, however, which is conspicuous throughout the new growth and the suckers from the same plant (which, of course, are really new growth from the same plant.) This type appears to be the result of a genetic abnormality.

CRUMBLE BERRY was found to be causing very severe losses in black raspberries in the Chehalem Mountain area of Oregon. Plants were vigorous and had produced large numbers of flower clusters, but in each flower only a few ovules had been fertilized and each fruit consisted of from two to six normal drupelets with the others dry and undeveloped. All varieties of black raspberries are affected. Application of various minor elements to the soil has not reduced the losses caused by the trouble.

DEAD ARM, a loss of fruiting laterals, was seen in the Washington red raspberry in Whatcom County, Washington and in Loganberry in Marion County, Oregon. Affected fruit spurs are normal in appearance except that they are frequently smaller than usual. As the fruit approaches maturity any appreciable movement of the spurs causes them to snap off. Apparently the weight of the fruit or movement due to the winds snaps off many of the spurs. There is no evidence of

insect injury or of decay in connection with the trouble.

LEAF SCORCH, characterized by the sudden wilting and necrosis of portions of the leaf was observed on Washington red raspberries in the vicinity of Puyallup, Washington.

ABNORMALITIES PROBABLY CAUSED BY UNSEASONABLY COLD WEATHER

Four types of abnormalities attributed to the unseasonably cold weather of early 1950 were seen throughout the Pacific Northwest on all varieties of red raspberries.

1. Lateral bud development on new canes has produced many branches in contrast with the usual straight unbranched canes.
2. A proliferation of fruit spurs. Instead of the customary single fruit spur from each leaf axil, from two to five weak fruit spurs were produced. Usually these multiple spurs were not more than 12 inches in length and produced only a few small berries. It is probable that the bud was killed by cold weather late in the spring and the fruit spurs developed from auxiliary buds which would otherwise have remained inactive.
3. A stippling along either side of the veins, somewhat resembling the "specking" caused by leaf-hopper feeding but confined to the veinal areas, was observed in red and black raspberries throughout the area. This is the same type of frost injury pictured in Michigan Technical Bulletin 80, 1927.
4. Deeply serrated leaves to which the term "fern leaf" might well be applied, were observed throughout the area on red raspberries.

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EFFECT OF ISOPROPANOL, AS A CONSTITUENT OF A GLYOXALIDINE FUNGICIDE, ON THE RIPENING OF BARTLETT PEARS AFTER HARVEST

E. E. Wilson

Mature but unripe Bartlett pears were utilized in tests to determine the lethal effects of various chemical preparations on Erwinia amylovora. The fruit were dipped into the preparations, placed in glass containers, and held at 70° to 75° F. At such temperatures, the untreated (water-dipped) fruit ripened within seven to eight days. It was noted, however, that fruit treated with a preparation containing 1/4 percent of the glyoxalidine formulation known as 341C changed from a green to a yellow color, became soft, and, judging from taste, were fully ripe within four or five days. Inasmuch as the 341C formulation contained 32.8 percent isopropanol in addition to the glyoxalidine, it was desirable to determine whether one or both constituents were responsible for the observed effect. Consequently, in further trials preparations of 1/4 and 1/8 percent 341C were compared with a preparation containing 1/12 percent isopropanol. After being treated, the fruit were placed in open wooden boxes and stored at 70° F. Through the cooperation of Dr. L. L. Claypool of the Pomology Division, the Magness-Taylor pressure-tester (with pear head) was used to determine ripeness of representative samples of the fruit at daily intervals. No difference in rate of ripening was found in one lot of Hardy pears which proved to be partially ripe when treated. With Bartlett pears, however, the following were average pressures for six successive days after treatment:

	Days after treatment					
	1	2	3	4	5	6
Control	13.9	11.15	7.00	4.43	3.01	1.94
1/4 percent 341C	13.8	10.80	5.35	3.50	2.23	1.83
1/8 percent 341C	15.3	11.05	6.35	2.83	2.51	1.83
1/12 percent isopropanol	13.8	8.30	6.50	3.07	2.17	1.83

Judging from these results, isopropanol was the constituent of 341C responsible for the increase in the rate of ripening.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA AGRICULTURAL EXPERIMENT STATION, DAVIS

AN UNUSUAL OCCURRENCE OF SCLEROTIA OF SCLEROTINIA SPP. WITH SEED OF CENTAUREA CYANUS

Kenneth F. Baker and Lily H. Davis

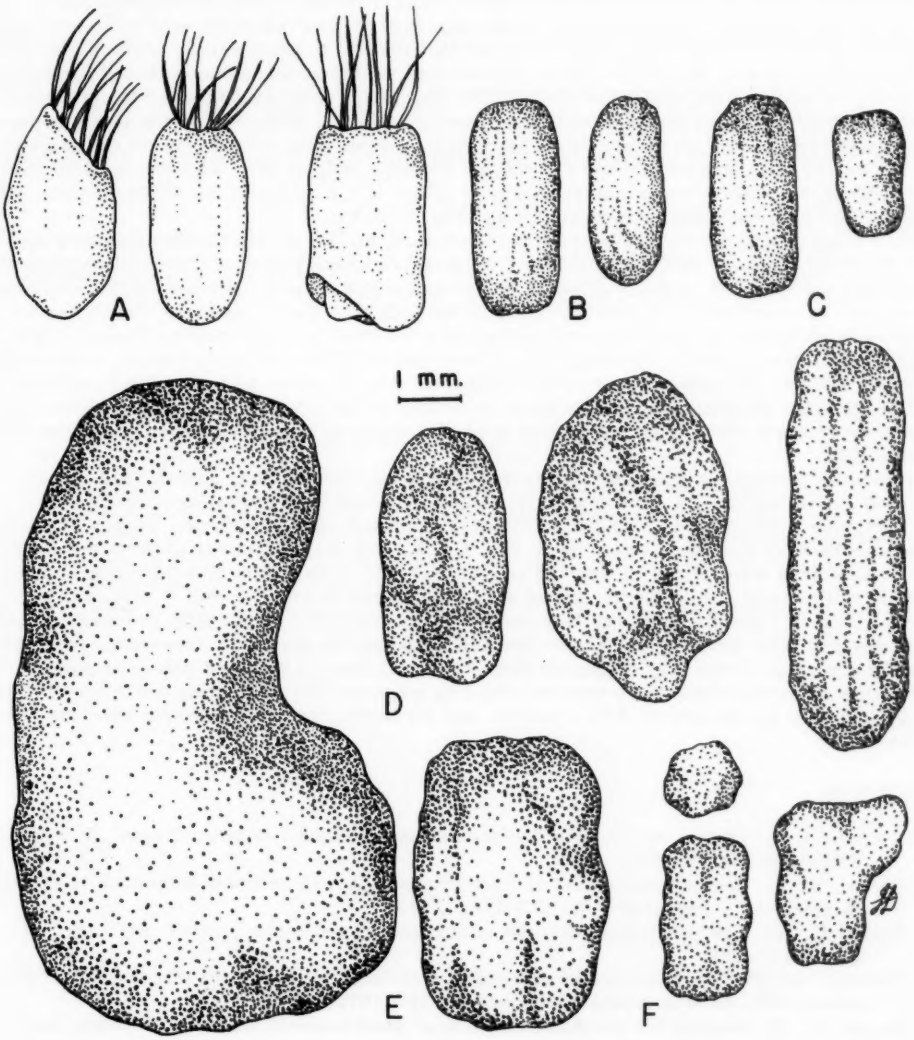

FIGURE 1. Camera lucida drawings of sclerotia of Sclerotinia spp. associated with seed of Centaurea cyanus. A. Three views of seeds of C. cyanus. B. Two typical sclerotia of S. minor from 1945 seed after it has been passed over a gravity separator. C. Two typical sclerotia of S. sclerotiorum from a similar 1945 seed lot. D. Sclerotia separated from a 1950 seed lot by means of a gravity separator. E. Two typical sclerotia of S. sclerotiorum produced by C in culture. F. Three typical sclerotia of S. minor produced by B in culture.

A sample of seed of Centaurea cyanus L. from a 1945 California commercial crop was received for examination in February 1946. Mixed with the tufted achenes (the "seed" of commerce) were a few black sclerotia of the same size and shape as the seed (Fig. 1, A, B, C). Some of

these, dipped in sodium hypochlorite and planted on agar plates, developed cultures of Sclerotinia sclerotiorum (Lib.) de Bary. Others, indistinguishable from these sclerotia in appearance, produced colonies of S. minor Jagger. About 20 percent of the sclerotia of these fungi grew when planted on agar after being kept nearly 4 1/2 years under dry laboratory conditions.

Apothecia of the two fungi developed in a cool greenhouse in flats of pasteurized soil sowed with these seed lots. Ascospores that were discharged from the apothecia infected stems of the bachelor button, demonstrating effective transmission of these organisms with Centaurea seed.

Such transmission was made possible by the fact that the sclerotia were of the same shape and size as the seed and, therefore, were not removed by the screens and cleaning machinery. Examination of some of the cleanings of the 1950 crop after being passed over the gravity separator showed that sclerotia differing from seed in size and shape were effectively separated by that machinery. Although most of the sclerotia thus separated out were about the same diameter but longer than seed, some were irregular in shape (Fig. 1, D). The sclerotia formed by the two fungi in host tissue were indistinguishable (Fig. 1, B, C) despite the characteristic differences exhibited by them when formed in culture (Fig. 1, E, F).

These fungi commonly invade the pith of infected stems and produce sclerotia there that conform in size and shape to the central cavity. In base-branching plants of Centaurea cyanus the many slender stems are frequently infected and produce sclerotia of the shape mentioned here. The irregular sclerotia probably are produced in the pith of large stems or in flower heads, and the stems are shattered in threshing, releasing the sclerotia, as suggested by Pape (12) and Bisby (3). The dense growth of these plants in seed fields favors moisture retention following the frequent ditch irrigations. Much of the Centaurea seed is grown along the coast of California where the fogs also maintain moist conditions favorable to the fungus. Sclerotia of these pathogens apparently occur commonly mixed with seed of Centaurea cyanus produced under the conditions described.

Sclerotia of Sclerotinia sclerotiorum mixed with seed of bean, pea, radish, red clover, white clover, crimson clover, Scorzonera hispanica L., sunflower, Medicago lupulina L., alfalfa, Anthyllis vulneraria L., white sweetclover, soybean, dahlia, carrot, pepper, and Jerusalem artichoke have also been reported (1, 2, 3, 4, 5, 7, 9, 12, 15). Sclerotia of S. minor have been reported mixed with seed of red clover and peanut (8, 10, 11). Sclerotia of Sclerotinia spermophila Noble and S. trifoliorum Eriks. occur with seed of red clover (10, 14), S. ricini Godfrey with castor bean (6), and S. porri van Beyma with leek seed (13). Apparently in most of these instances the sclerotia were not the same size and shape as the seed and, therefore, could be removed by cleaning. Pape (12) suggested removal by (a) dusting the seed with iron powder that adhered to the sclerotia which were then removed by magnets, (b) by floating out sclerotia and defective seed on a 23-24 percent KCl solution, and (c) treatment with mercuric chloride 1-1000 for 30 minutes.

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DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, LOS ANGELES 24

OCCURRENCE OF CROWN GALL ON YARROW

Peter A. Ark

Crown gall has been observed on ornamental types of yarrow imported from out of the State. Out of the four varieties of yarrow in a large consignment two kinds, namely, Achillea ptarmica and A. ptarmica var. Snow Ball, were the most affected. Two other varieties, A. filipendula and A. ptarmica var. Boule de Neige were practically free of the galls. The affected plants had a large number of galls on the fibrous roots. Large galls occurred under the crown of the plants. The affected plants did not seem to have suffered serious injury from the disease and when observed a year after planting were thrifty. Agrobacterium tumefaciens isolated from soft galls on the roots was pathogenic on tomato, sunflower, peach, Achillea filipendula, and A. ptarmica grown in the greenhouse.

Greenhouse tests showed A. ptarmica to be very susceptible to the pathogen. The disease is easily induced on any part of the plant and a sizable gall can be observed five days after puncturing the stem with a contaminated needle. This plant proved to be very useful for teaching purposes since the plants are easily propagated from cuttings and many shoots can be produced in a plant in one pot. The plant can stand much abuse when not in use and responds promptly to reasonable care. In teaching plant pathology it can easily be substituted for such standard crown gall test plants as tomato, sunflower, and Bryophyllum.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

BACTERIAL DISEASES OF ORCHIDS

Peter A. Ark and Mortimer P. Starr

There are three bacterial diseases of orchids in central California which sometimes cause considerable losses. A fourth bacterial disease, although extremely destructive, is of rare occurrence and so far has been observed only once.

Brown spot of Phalaenopsis and Cattleya, caused by Phytomonas cattleyae, is common in the greenhouses (Ark and Thomas (1)). Lately it has been especially severe in Phalaenopsis seedlings in community pots. Symptoms on small Phalaenopsis seedlings consist of one or more circular water-soaked spots of dark green color at first which changes into dirty green through various shades of brown and finally black. The disease when unchecked spreads rapidly resulting in great numbers of dead seedlings. In one orchid house large old plants of Phalaenopsis were severely attacked and considerable losses were inflicted before the disease was stopped. On large plants of Phalaenopsis the disease can start at any place on the blade, finally reaching the growing point. There is present a considerable exudation of the infectious material on the surface of the diseased part which serves as a potential source of inoculum to be picked up by the water during syringing of the plants and thus carried to other plants. The disease starts through the stomata on young plants and through various wounds on full-grown plants.

On Cattleya the disease does not seem to move as fast as in the Phalaenopsis and is limited to the older leaves. The lesions are more discrete and well delimited. Often the lesions are sunken and black. The mode of infection in Cattleya is the same as in the Phalaenopsis. The spots on Cattleya are checked by local application of corrosive sublimate, 1:1000, applied with a cotton swab. The treatment has to be repeated several times until the disease is stopped.

The disease can be prevented to a great extent by avoiding overhead irrigation and giving good aeration. The seedling phase of the disease in Phalaenopsis is controlled by drenching with one of the following chemicals: 8-quinolinol benzoate (or sulfate), 1:2000, or the sodium salt of o-hydroxydiphenyl, 1:2000. If desired, the pots having a community of seedlings can be submerged in the solution and left an hour or longer. The treatment can be repeated. No injury has been observed on experimental plants so treated and many growers are using this method of control. The treatment can be repeated in about two weeks, if necessary. The chemicals mentioned above do not cause any residual effect since they are broken down by bacteria in the substratum. Large Phalaenopsis plants should be treated by submerging them in 8-quinolinol benzoate (or sulfate) or the sodium salt of o-hydroxydiphenyl (Natriphene) for several hours. By this method seriously infected plants 30 years of age and older were completely cured.

Brown rot of Cypripedium was found to be very serious in several houses. The disease starts as a small circular or oval water-soaked and somewhat greasy light brown spot. Several spots may occur on one leaf. The lesion becomes somewhat sunken, dark brown and later deep

chestnut in color, and the margin is well defined. The infection works its way into the growing point at a very rapid pace if the temperature in the house is above 65° F. and the humidity is in the neighborhood of 70 percent or higher. Affected crowns shrivel and the leaves droop. The disease seems to proceed from the periphery of the plant inward. The organism isolated from the lesions resembles Erwinia cypripedii and produced the disease upon being inoculated into healthy plants, The disease is controlled by the same methods described above.

A disease of Miltonia characterized by leaf scorch and pseudobulb rot has been found to be caused by bacteria. A bacterium was isolated from early stages of the disease and Koch's postulates fulfilled. The taxonomic position of the pathogen has not yet been determined. The disease produces tip blight or scorch which may be arrested and then appear toward the base of the leaf in the form of a water-soaked streak leading into the growing point on the pseudobulb. The infected pseudobulb tissue first becomes straw yellow and later orange red or bright red. Rotting of the pseudobulb is accomplished by other organisms which sometimes follow the primary pathogen. The pathogen progresses through the rhizome of the plant and can attack new pseudobulbs from below the crown. Attacked leaves frequently drop and only the orange-red pseudobulb may remain in the pot. The infection starts from wounds, which may be numerous on the brittle leaves of Miltonia. The disease spreads from plant to plant if segregation and chemical treatment are not followed. 8-quinolinol benzoate and Natriphene gave the best results in checking the disease.

One case of soft rot of Cattleya orchids caused by Erwinia carotovora as described by Limber and Friedman (2) has been found in California.

SUMMARY

Brown spot of Phalaenopsis and Cattleya orchids caused by Phytomonas cattleyae, brown rot of Cypripedium caused by Erwinia cypripedii, and leaf scorch and pseudobulb rot of Miltonia caused by an unidentified species of bacterium are frequently found in orchid houses in central California and can lead to considerable losses. The soft rot of Cattleya, caused by Erwinia carotovora, has been found only once.

Bacterial diseases of Phalaenopsis, Miltonia and Cypripedium can be controlled by drenching or soaking in 8-quinolinol benzoate (or sulfate) and the sodium salt of o-hydroxydiphenyl (Natriphene). Brown spot on Cattleya is controlled by local application of 1:1000 corrosive sublimate.

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DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA
DIVISION OF BACTERIOLOGY, UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA

BLACK SPOT OF VANDA ORCHIDS

Peter A. Ark and William C. Snyder

Recently flower spoilage has been observed on blooms of Vanda tricolor, Lande, shipped from Hawaii to San Francisco for re-distribution to various parts of the country. Flowers received on both the Chicago and San Francisco markets have shown petal discoloration and black spots of varying sizes in the flower throat below the labellum, rendering the blossoms unsalable. Under conditions of high humidity the blackened areas enlarge. After five days in a moist chamber affected flowers may become entirely black, at which time a dark grey fungus growth appears on the discolored flower parts.

Isolations made from spoiled flowers have yielded a Glomerella which in pure culture on natural media, consisting of sweetpea straw and water agar, has produced a Colletotrichum stage. Artificial inoculations made by spraying healthy flowers with this fungus have resulted in the same type of spoilage as that which occurs on the imported flowers. Although Glomerella is not uncommonly found on orchid plants, it does not appear to have been recognized as an ecomomic

factor on Vanda blossoms.

Control of the black spot has been effected by spraying the blossoms with a solution of Bioquin 700 (o-quinolinol benzoate) before the appearance of symptoms. No injury has been observed on sprayed flowers shipped by air from San Francisco to Chicago for delivery to retail florists.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

SODIUM SALT OF O-HYDROXYDIPHENYL, A PROMISING CHEMOTHERAPEUTANT

Peter A. Ark

Stoddard and Zentmyer reported good results from the use of an organic compound known as 8-quinolinol benzoate against damping-off (1). The writer has found this chemical useful in controlling various fungus and bacterial diseases of orchids. During the last two years the writer has employed another organic compound, the sodium salt of o-hydroxydiphenyl, which merits investigation as a promising agent for the control of damping-off fungi and certain bacterial pathogens.

This compound has been known to the medical profession and sold under the trade name Natriphene. It is used in treating mycotic skin diseases as well as for general hygienic purposes. The chemical is not stable and is denatured by light and strong alkali. In vitro tests indicate its high degree of toxicity against many fungi and bacteria.

To evaluate its effectiveness against damping-off caused by Rhizoctonia solani tomatoes, peppers, marigolds, snapdragons, and orchids were used. With the exception of the orchids, 100 seeds of test plants were sown in small flats of pasteurized soil, each test consisting of three series. In one series, Rhizoctonia solani, previously isolated from the corresponding plants, was established in flats of pasteurized soil by introducing the fungus mycelium two weeks before planting the seed. The second series was similar except that as soon as germination was observed the flats with the fungus were saturated with a 1 to 1000 solution of sodium salt of o-hydroxydiphenyl to one of the Rhizoctonia-infested soils. In a third series the plants were grown in pasteurized soil alone. Counts of diseased plants were made weekly for a month. A typical protocol of the results is shown in Table 1.

Table 1. Effect of Natriphene treatment on damping-off caused by Rhizoctonia. Number of plants at the end of 30 days.

Plant	Pasteurized soil	Rhizoctonia added to the soil	4 applications of Natriphene to soil containing Rhizoctonia
Tomato	90	20	75
Pepper	85	0	78
Marigold	100	5	95
Snapdragon	98	0	95

A few nurserymen, growing annuals for sale as bedding plants, used this chemical as indicated above and obtained similar results. In the course of the investigation it was observed that occasionally slight burn was produced on very tender plants when a technical grade was used. The medicinal grade has not given any injurious effects.

This chemical was used successfully to control certain bacterial diseases of orchids. The method consisted in complete immersion of diseased plants in a 1:2000 solution of the chemical for 60 minutes or longer, There was no visible injury on treated plants.

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DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

BOTRYTIS SPOT AND BLIGHT OF TUBEROSES IN CALIFORNIA

Peter A. Ark and Neil A. MacLean

Commercial plantings of tuberoses (Polianthes tuberosa L.) in coastal California grown for cut flowers and bulbs frequently develop severe spotting and blighting of the foliage and the flowers coincident with overhead watering. In some years the foliage may be blighted completely and there may even be a rot of the neck of the bulb. The flowers spotted by the fungus become unsalable and this causes considerable loss to the grower.

In the early stages of the disease there develop very small dark circular to irregularly shaped spots which at high humidity enlarge and become round with concentric markings and light to dark brown color (Fig. 1). When the spots coalesce streaks can be observed on the leaves. Tips are usually blighted. Flowers show spotting and blighting. The disease attacks sepals and the flower stalks.

FIGURE 1. Botrytis spot and blight of tuberoses.

Plating of the spots from both the flowers and the leaves consistently yielded a fungus which was determined as Botrytis elliptica (Berk.) Cke. The spore measurements fall in the lower grouping as given by Westerdijk and van Beyma (4). This is another record of Botrytis elliptica occurring in a non-lily host. Previous records (1, 2, 3) have shown the fungus to be the cause of leaf spotting on Colchicum autumnale L., Gladiolus spp. and Erythronium grandiflorum Pursh. var. pallidum St. John.

Spraying healthy greenhouse plants grown in pasteurized soil with conidia of the fungus isolated from the lesions produced spots identical in all respects with those from the field. The fungus was successfully reisolated from the artificially inoculated plants.

The disease thrives under the conditions of high humidity and cool summer temperatures

prevailing on the coast near Half Moon Bay, California. Spread of the disease is facilitated by the sprinkling system of irrigation. The disease is frequently carried in the bulb in cases where the neck of the bulb has been invaded. In this case small irregular sunken spots may be found on the bulb and the organism may be isolated from the surface of the lesion.

To prove the transmission of the disease by bulbs, 100 tuberose bulbs selected for the neck lesions, and 100 healthy bulbs were planted in the field, well separated from the main field. The two experimental lots were also separated from each other by a distance of 12 feet. Counts on diseased plants were made in the middle of July and it was found that bulbs which had neck lesions gave 100 percent diseased plants, while in the apparently healthy check a few lesions were found on the leaves of ten plants and the disease was not severe. The following season the bulbs from a large diseased field were dipped, before planting, in (a) 1/2 percent Lysol solution for 60 minutes, and (b) in 1:2000 solution of Natriphene, sodium salt of o-hydroxydiphenyl for three hours. The treated bulbs were planted in prepared ground immediately after the treatment. A check of 500 untreated bulbs was planted in a separate field some distance away from the treated plot. The results were taken at the end of July and in September. The number of infected plants in the Lysol treated lot was 7 percent, in the sodium salt of o-hydroxydiphenyl treated lot 3 percent, and in the untreated check 85 percent. It appears that plantings from bulbs treated either with Lysol or sodium salt of o-hydroxydiphenyl show very little disease in comparison with the check and that the above treatments can be recommended as control measures for this trouble.

To check the spread of the disease in the field when it had not caused serious damage to the leaves, spraying the plants with (a) ammoniacal copper, 2 gal. per 100 gal. water, (b) Greenol, and (c) sodium salt of o-hydroxydiphenyl, 1:2000, held the disease under control for a maximum of two weeks after which the treatment had to be repeated.

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DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

PHYTOPHTHORA ROT OF PEPEROMIA

Peter A. Ark and Thomas A. DeWolfe

Peperomia plants are extensively propagated by nurserymen in the San Francisco Bay area. These herbaceous, fleshy succulent plants are sold as foliage plants and the demand for them at certain times of the year is heavy. The species and varieties most commonly used by the propagators include: Peperomia maculosa, P. minima, P. rotundifolia, and P. sandersii. Propagation is usually by stem cuttings or by leaves inserted either in straight sand or in a mixture of sand and vermiculite.

A serious disease in Peperomia plants was observed recently. In one greenhouse 50 percent of the plants was affected by the disease. The disease occurs on all parts of the plant. On large plants the first symptom consists in a pronounced browning at the ground line. This browning may be either one-sided or encircling the stem. Soon the lower leaves droop and with the further progress of the disease the whole plant wilts. On very small plants infection seems to start from the blade of the leaf touching the soil and to progress into the stem, causing girdling of the plant and subsequent death. In the cutting box the disease affects the base of the plant and if the cuttings are rooted the roots can be involved first.

Tissue cultures of diseased parts on potato-dextrose agar and insertion of the diseased tissues into an apple yielded a pure culture of a water mold. This fungus grown on boiled hemp

seed produced in three days an abundance of swarm spores. These were used to inoculate steam sterilized soil in which healthy Peperomia plants were growing, and also to inoculate leaves of healthy plants. All these inoculations produced typical symptoms of the disease. The original fungus was reisolated from artificially inoculated plants. All species and varieties of Peperomia tested were equally susceptible to the fungus.

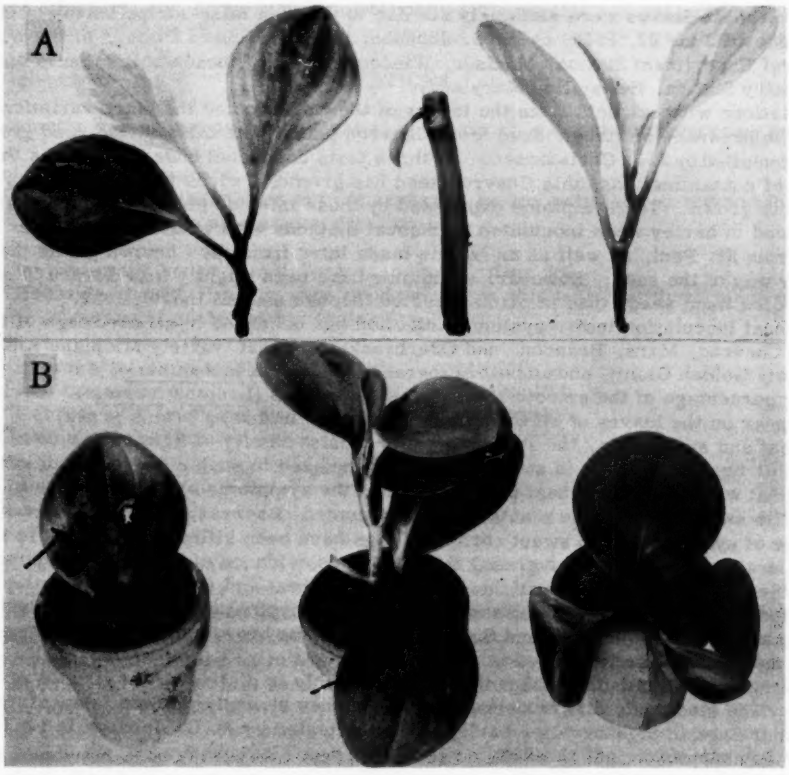

FIGURE 1. Phytophthora rot of Peperomia. A. Discoloration of the stems. Natural infection. B. Artificial inoculations with zoospores of Phytophthora palmivora.

The causal fungus was determined by Dr. John T. Middleton to be very close to Phytophthora palmivora.

The disease was controlled in the two large foliage plant establishments by using cuttings from known healthy plants and planting them in a pasteurized soil mixture. Steamed sand used in clean propagation boxes yielded healthy Peperomia cuttings.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY AND RIVERSIDE

A SEED-BORNE VIRUS CAUSING FALSE-STRIPE SYMPTOMS IN BARLEY

H. H. McKinney

Leaves of Chevron and of Mars barley, showing false stripe in the nursery at St. Paul, Minnesota, were sent to the writer by G. A. Wiebe and by Dick Pon, in July and August 1950. The signs in these leaves were strikingly similar to those in false-stripe leaves of Chevalier barley collected June 27, 1913, by A. G. Johnson, in the "Orchard Plats", of the Wisconsin Agricultural Experiment Station, Madison, Wisconsin. The Johnson collection is on file at the Plant Industry Station, Beltsville, Maryland.

Inoculations with extracts from the leaves of the Chevron and the Mars varieties, have revealed the presence of virus. Seed from Chevron plants with false stripe, and grown at St. Paul, was supplied by J. J. Christensen. In three tests conducted under conditions that eliminate likelihood of contaminations, this Chevron seed has given 132 virus infected plants out of a total of 226 plants grown. The symptoms expressed by these infected plants are comparable with those induced in barley when inoculated by manual methods with virus isolates from the leaves received from St. Paul, as well as an isolate made later from the Chevron plants that became infected by way of the seed. However, symptoms have been slightly less severe in most of the plants infected from seed, than in plants infected through manual inoculations.

In manual inoculation tests, systemic infection has occurred in all seedlings of barley (varieties Chevron, Mars, Brandon, and Oderbrucker), wheat (variety Michigan Amber), sweet corn (variety Golden Giant), and smooth crabgrass (Digitaria ischaemum), but it has occurred in a very low percentage of the smooth brome grass seedlings (Bromus inermis). Local lesions usually appear on the leaves of all inoculated seedlings, including brome grass.

In wheat and sweet corn, the systemic signs consist chiefly of light yellow or bleached, continuous or broken stripes, in some cases being massed to such a degree as to render the leaves almost white. As the wheat plants develop, the symptoms are less severe in the new leaves. With sweet corn, some plants are badly stunted, whereas, others may produce new foliage free of symptoms. No sweet corn seedlings have been killed by the virus in these tests. In barley, crabgrass, and brome grass, there is more evidence of brown necrosis with the yellow or bleached patterns, than is the case with the wheat and sweet corn varieties studied. In barley and crabgrass, the brown necrotic stripes are typical of false stripe; however, the chlorotic markings predominate, and these, as well as the brown markings, are regarded as signs of a mosaic disease.

Preliminary studies indicate that this virus probably is distinct from viruses previously described. The circumstantial evidence suggests rather strongly that the false-stripe and the chlorotic markings in the Chevalier barley plants collected by A. G. Johnson in 1913 were induced by this virus.

DIVISION OF CEREAL CROPS AND DISEASES, U. S. DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

COMMERCIAL CEREAL SEED TREATMENT WITH PANOGEN: COMMENTS AND EVIDENCE ON SOME PUBLISHED OBSERVATIONS

Martin Tveit

Koehler and Bever's article "Oat and Wheat Seed Treatments in 1950 in Relation to Dosage, Storage before Planting, and Thoroughness of Mixing" (PDR 34 (9): 259-261) contains certain statements on which we wish to comment and to present some of our own experimental evidence.

QUOTATIONS

The statements are quoted as they appeared in the paper under their respective headings.

In the introductory paragraph, page 259:

> "Unfortunately, there does not yet appear to be any satisfactory method for applying Panogen on a commercial scale."

In the first paragraph under "Smut Control in Oats", page 260:

> "Treatments with Panogen gave good results in this test, but those made on a commercial scale gave poor results."

Under "Commercial Treatments with Panogen", page 260:

"Random sample	92.7% germination
"Kernels with least red dye	98.0% germination
"Kernels with most red dye	23.2% germination

"It appeared obvious that the mixing was inadequate ....."

COMMENT

Different harvesting equipment may account for the fact that the percentage germination quoted from these authors differs from that obtained in our own test described below. As to the uneven distribution of color in relation to germination, we feel that no direct conclusion can be drawn, because in all seed lots a certain percentage of mechanically injured and abnormal seed occurs, which in many replicated tests has been shown to absorb more of the color material from the Panogen liquid than normal seed does. This abnormal seed, moreover, would only give a poor germination, and in tests in which Panogen has been used with or without mercury we have found that the strongly colored seeds give about the same percentage of germination in both cases. Such abnormal seed germinates approximately as stated in the paper, but as, in general, it represents only 2 to 5 percent, or on certain occasions perhaps a bit more, of the total, this seed will not make a great difference in the percentage germination of the seed lots as a whole.

EXPERIMENTAL EVIDENCE

In a new regular Panogen machine, J. S. type, capacity two tons per hour, mercury-free Panogen at the rate of 3/4 oz. per bushel was used to treat wheat samples, then regular Panogen at the same rate was used to treat samples from the same seed lots. A sample was taken every minute for 20 minutes. From each treated sample 1000 seeds were taken. The samples were divided into three groups according to color: strongly colored, moderately colored, and weakly colored or not colored. Germination tests were made from each group by the standard method on filter paper and the results obtained are recorded in the Table as an average of the 20 samples taken.

	Panogen 2.1% Hg		Panogen Hg free	
	Percent kernels divided by color	Percentage germination	Percent kernels divided by color	Percentage germination
Check - untreated	--	69	--	69
Strongly colored	1.52	66	1.54	67
Moderately colored	7.04	78	6.91	67
Weakly colored	91.44	83	91.55	73
Random sample	--	80	--	71

These figures and many other similar tests have made us quite confident in our interpretation of the problem discussed above.

It might be stated, however, that the aim of the color is only to guide those who handle the grain in distinguishing thereby the treated lots of seed from untreated ones.

CONCLUSION

Research over a ten-year period has been conducted to clarify the effect, action, and spreading of mercurial liquid seed disinfectants, and information about these tests are available in our Panogen stencil 10-50.

As to the poor smut control obtained with commercially treated seed, we do not have any comments and we do not know the writer's results. However, as to generality of the smut control, we should like to stress the point that from the many million bushels of seed that were treated in the United States and in Canada in 1950, no claims of poor smut control have been made.

LAUXEIN-CASCO PHYTOPATHOLOGICAL LABORATORY, PANOGEN, INC., NEW YORK, NEW YORK

DIPLODIA CROWN ROT OF ALFALFA

Bailey Sleeth

Crown rot of alfalfa is a common and serious disease in the irrigated areas of the Southwest. Losses from crown rot are greatest in stands three to four years old or older. However, severe losses have been observed in small localized areas in stands 9 to 12 months old on the Yuma Mesa in the southwest corner of Arizona. It has been necessary to renovate and reseed a considerable acreage of three-year-old alfalfa on the Yuma Mesa, because of thinned out stands caused by crown rot. The losses from crown rot are most noticeable in August, September and October in the Yuma area.

There is considerable evidence that crown rot of alfalfa may be caused by more than one pathogen, and that predisposing factors play no small part in the crown rot complex. Because of five to seven cuttings made each year on the Yuma Mesa the chances for mechanical injury to crowns are high. Also, depletion of food reserves resulting from the frequent cuttings may be an important factor. Brown and Streets (1) report a Fusarium sp. as a probable pathogen and certain management practices as contributing factors. In New Mexico Fusarium solani has been reported by Staten and Leyendecker (2) as a cause of crown rot as well as a root rot of alfalfa. In the Yuma Mesa area Fusarium spp. have been the most commonly isolated fungi from crown rotted plants, and less frequently isolated has been a Diplodia sp.

In the early fall of 1948 several dead and dying alfalfa plants affected with crown rot were found with Diplodia gossypina fruiting on the dead crowns (Figure 1, A) and the upper part of the taproot in stands three and four years old. There has been a considerable increase in the number of such plants in the same fields in the past two years. From these plants pure cultures of Diplodia were obtained from the margin of incipient decay as well as from the more advanced and typical decay. Also, cultures of the fungus were obtained from dying plants with typical dark to black crown rot (Figure 1, B) but on which no pycnidia had developed.

The pathogenicity of the Diplodia was demonstrated by inoculating healthy two-year-old alfalfa plants on September 18. Eight days later the fungus was reisolated one to three inches beyond the point of inoculation. Typical crown rot symptoms had developed. In some instances pycnidia (Figure 1, C) had developed on the recently killed crown of the inoculated plants. No crown rot developed in the controls. The inoculations were made by inserting a bit of mycelium from a test tube culture into a knife wound in the crown of the plants.

Diplodia crown rot can be distinguished in the advanced stages by the black rot (Figure 1, B) which develops in the crown and rapidly advances into the taproot. Later typical black erumptent pycnidia develop. The rot might be characterized as dry; however, following irrigation and the invasion of secondary organisms the rot usually becomes soft and soggy. Diplodia has been isolated at the crown and from the taproot six inches below the soil surface. The fungus can advance rapidly once a plant is infected, as shown by inoculation where it had penetrated three inches within eight days after being inoculated.

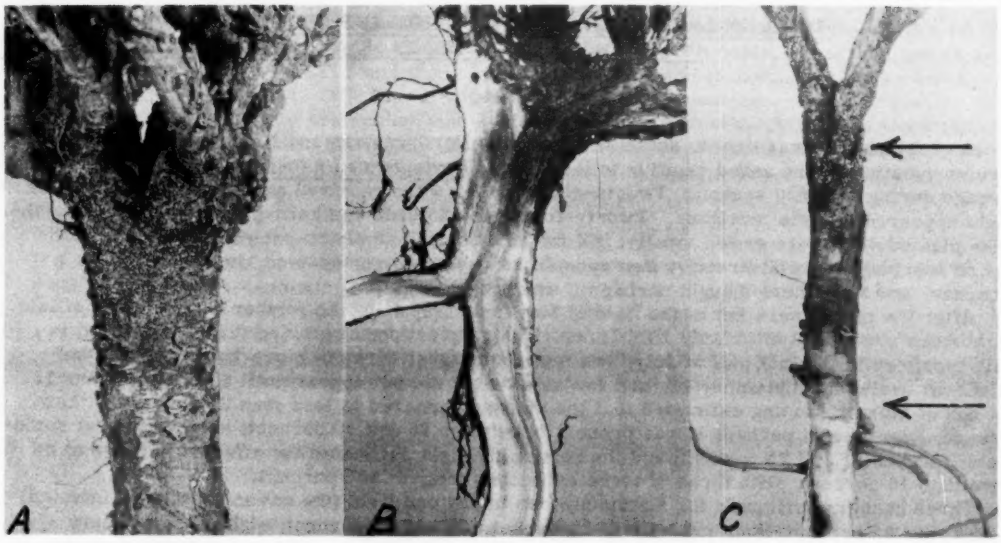

FIGURE 1. Diplodia crown rot of alfalfa. A. A 4-year-old plant killed by Diplodia gossypina. Pycnidia have formed on the recently killed crown. B. A split crown with typical black Diplodia rot at the crown and discoloration extending into the taproot. C. Eight days after inoculating a 2-year-old alfalfa plant pycnidia had developed near point of inoculation (upper arrow) and decay had progressed two inches down the taproot (lower arrow).

On the basis of microscopic and cultural observations the fungus fits sufficiently well the description of Diplodia gossypina Cke. (D. natalensis Pole-Evans) as reported by Verrall (4) and Stevens (3) to be so designated. In culture the fungus grows rapidly at high temperatures, 27 to 35 mm. in 24 hours. The average growth rate in culture for 8 isolates in 24 hours was 21 mm. at 25° C. and 30 mm. at 30° C. At high temperatures the isolates tend to produce a reddish coloration in the agar and mycelial mat instead of the usual dark gray to black shades produced at room temperatures. Mature pycnospores produced in culture are dark, 1-septate with longitudinal striations and mostly 23 to 26 x 13 microns in size. Immature spores may be non-septate and colorless.

D. gossypina is at present of minor importance as a cause of crown rot in alfalfa. Its future importance, increase in severity in the Yuma area, and spread to other areas will no doubt depend upon a number of factors. Cultural operations which cause a great amount of crown injury at the time when there is an abundance of viable inoculum and the subsequent buildup of inoculum from whatever source will affect in a great measure losses that may be expected from Diplodia crown rot. In the case of the Yuma Mesa area the proximity of citrus groves may be an important factor as a source of inoculum. D. gossypina is an important and widely distributed citrus pathogen; however, its prevalence or importance in the citrus groves of the Yuma area is undetermined.

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DATA ON LATE BLIGHT DISTRIBUTION AND SEVERITY IN NORTHWESTERN OHIO IN 1950

J. D. Wilson

A questionnaire was sent to about 40 canners in Northwestern and Northern Ohio in which various questions were asked relative to late blight (Phytophthora infestans) occurrence in their acreage during the 1950 season. Twenty-eight replies were received and all reported that late blight appeared in their territory. Twenty-five of the 28 used southern-grown plants -- the other three planted only those grown locally. Of the 25 who used southern-grown plants, 21 stated that no late blight was observed in the transplants as they were received, two reported its presence, and two others thought perhaps it was present.

After the plants were set in the field in May and early June, no further observations of late blight were made until after July 15. Three of those questioned reported that it was found in their territory in the last half of July, two more by August 15, nine more by August 31, an additional twelve by September 15, and two said that it did not appear until after September 15.

Six of those reporting estimated that the disease appeared in less than 25 percent of their acreage; four thought perhaps it was present in between 26 and 50 percent; another ten, at somewhere between 51 and 75 percent; and the remaining eight estimated the affected acreage to be more than 75 percent, with three of these estimates being at 100 percent.

Three canners estimated the maximum loss in any one field (the one most heavily infected) at less than 25 percent, four judged it to be between 25 and 50 percent; eight at somewhere between 51 and 75 percent; and thirteen reported a maximum loss of 75 percent or more, of which eight observed fields where the loss was 100 percent.

Answers to the question concerning the percentage of total acreage treated indicated that approximately 62 percent was sprayed or dusted one or more times. Approximately 60 percent of the acerage showed the disease. Apparently control measures were not effective in at least one-third of the acreage treated. As a matter of fact, the disease apparently was so severe in some fields that it would not be reasonable to expect that complete control could have been obtained by any means. However, most of those who replied indicated that the disease was much less severe in treated fields.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OHIO AGRICULTURAL EXPERIMENT STATION, WOOSTER

PRESERVATION OF THE SPORES OF THE CUCURBIT DOWNY MILDEW FUNGUS BY FREEZING DETACHED LEAVES

G. K. Parris

Research with the downy mildew fungus of cucurbits, Pseudoperonospora cubensis (Berk. & Curt.) Rostow., has been handicapped by a lack of a constant supply of spores of the fungus. Being an obligate parasite, its spores must be collected from infected cucurbit plants, either in the field or in the greenhouse. Greenhouse plants are not a satisfactory source, for spores are produced best at high humidities. Sooner or later an investigator working with this fungus in the greenhouse finds himself without conidia to perform inoculations. This is particularly disturbing to workers in northern climates, and plant breeders must wait until summer to obtain readings on the resistance or susceptibility of their genetic material. Even in Central Florida it can be difficult to find conidia out-of-doors during November through March. If frosts come early, all cucurbits are dead by November 15. In the Homestead section of the State it is possible to find infected cucurbits almost all year; this town, however, is 300 miles distant from Leesburg.

This note is written to report incomplete findings, which may serve as an aid to other investigators interested in the development of disease resistance to downy mildew of cucurbits.

Hamilton and Weaver (1) have reported that it is possible to preserve spores of Gymnosporangium juniperi-virginianae and of Venturia inaequalis by freezing at - 40° C. Galls of the rust fungus remained alive for 15 months, while sporidia in water could be kept for 3 weeks. Frozen conidia of V. inaequalis remained viable for 15 months. These workers mention that Dr. R. F. Suit had comparable results from freezing conidia of Plasmopara viticola, but give no detailed data.

It has been found possible to preserve the viability and infective power of the conidia of Pseudoperonospora cubensis, on detached squash leaves, for six months. Detached leaves are first frozen at - 30° to - 33° C., and then stored in a frozen-food locker at - 15° to - 18° C. (approximately 0° to 5° F.). The leaves were not enclosed in a container. After 6 months the leaves appeared completely desiccated, and conidia were plasmolysed. At 1, 3, 5, and 6 months' times the conidia appeared to be turgid. In further work now in progress, diseased detached leaves are enclosed in pliofilm containers, and frozen.

Literature cited

1. Hamilton, J. M. and L. O. Weaver. Freezing preservation of fungi and fungus spores. Phytopath. 33: 612-613. 1943.

WATERMELON AND GRAPE INVESTIGATIONS LABORATORY, FLORIDA AGRICULTURAL EXPERIMENT STATIONS, LEESBURG, FLORIDA

RUSSET MITE DAMAGE TO TOMATOES IN TEXAS IN 1950

P. A. Young, G. H. Godfrey, O. H. Calvert, and B. H. Richardson[1]

Russet mite damage to tomatoes has been found again in Texas[2], this time near Laredo, Eagle Pass, and Batesville in October and November, 1950. It was found again in Cherokee and Nacogdoches Counties, but was not serious in these counties as adequate control methods were used last year. The mites are very small (about 1/250 of an inch long) and cannot be seen without the aid of a magnifying glass. When the mites are very abundant, the symptoms of damage are prominent. Four large fields near Laredo and Eagle Pass were damaged severely. Because russet mites have become destructive in California, this report of their occurrence in Texas should serve as a warning to County Agents and growers in the tomato growing areas of Texas to watch for them and report them when they are found.

The symptoms of russet mite damage to tomatoes are striking and distinctive. They consist of browning of the epidermis of the main stem and branches, usually starting near the bottom or middle of the plant; the browning sometimes becoming shiny; prominent orange-yellow spotting on upper leaf surfaces related to heavy infestation on the lower surfaces; prominent browning and silvering of the lower leaf surfaces; downward curling of margins of leaflets with many mites; thickening of the leaf, with consequent brittleness; leaves dying progressively upward from the base of the plant and becoming brown or gray without shriveling. Flower clusters may be infested, causing typical discoloration of pedicels. Mites may be found in the flowers, and considerable shedding of flowers may occur; fruits may develop brown-checked corky peel. Yield of marketable fruits may be greatly decreased when small plants are attacked and the mites are not controlled.

Russet mites on tomatoes are easily controlled. They were controlled in the East Texas area in the spring of 1949 by sulfur dusting and early eradication of the plants after harvest. This decreased the population of mites so much that they were not found again until July, 1950, and then as mild infestations in only three fields of tomatoes.

The outbreaks of russet mites near Laredo, Eagle Pass, and Batesville were controlled quickly by sulfur dusting in 1950. Dusting the fields thoroughly with dusting sulfur at seven-day intervals for two or more applications proved adequate. Where other insects or diseases must be controlled at the same time, a fungicide or insecticide may be added to the dust, using at least 50% sulfur. Such a dust formula could be: sulfur 50%, DDT 5%, copper 5%, and Pyrax ABB 40%.

It is recommended that all of the tomato plants in a field be killed to destroy the russet mites and other pests as soon as possible after fruit picking has been completed. This method was very effective in Cherokee County in preventing damage from russet mites in 1950.

TEXAS AGRICULTURAL EXPERIMENT STATION, TOMATO DISEASE LABORATORY, JACKSONVILLE, TEXAS

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[1]Respectively Plant Pathologists of the Tomato Disease Laboratory at Jacksonville, Lower Rio Grande Valley Substation at Weslaco, Substation No. 19 at Winter Haven, and Entomologist at Substation No. 19 at Winter Haven.

[2]Young, P. A. A destructive disease of tomatoes caused by russet mites in East Texas. Plant Dis. Reptr. 33: 484-485. 1950. It cites earlier literature.

NEW RECORDS OR UNUSUAL OCCURRENCES OF PLANT DISEASES

NEW RACE OF WHEAT STEM RUST IN AUSTRALIA

By W. W. Waterhouse

The rust survey I started in 1921 is being carried forward. I feel that such knowledge is fundamental in the work of breeding resistant varieties.

At the end of 1948 a new race of wheat stem rust (Puccinia graminis var. tritici) turned up in southern Queensland and has now spread over the southeastern part of the continent where most of Australia's wheat is grown. It has completely altered the situation so far as stem-rust resistant varieties are concerned, but by using the continued back-cross system we have material coming forward which should meet requirements until the next major change takes place in the rust flora.

One would like to know a great deal more about the origin of these new races under our conditions: the barberry had nothing to do, I feel certain, with this latest change, and it really does not help greatly to conclude that mutation was responsible for it.

FACULTY OF AGRICULTURE, THE UNIVERSITY OF SYDNEY, SYDNEY, AUSTRALIA.

(From a letter to H. B. Humphrey, December 10, 1950. In transmitting this note to the Plant Disease Survey, Dr. Humphrey added the comment that "The airplane may be a factor in spore distribution.")

PINK ROT OF POTATO FOUND IN NEW YORK STATE

By C. W. Boothroyd

Several diseased potato tubers of the variety Katahdin were received in October from Orchard Park, New York. A brownish, rubbery rot was present at the stem end of most of the tubers, the diseased portion being separated from the healthy by a black margin. The lenticels and eyes were also blackened, and watery droplets exuded out of them when the affected area was gently squeezed.

The internally diseased portion of each tuber slowly turned to a bright pink and then to black after the potato had been cut open and exposed to the air for about 30 minutes. Oogonia and amphigynous antheridia were found in a few stolons and stems submitted with the tuber sample.

Isolations were made from diseased potato tissue near the advancing margin of the rot. A fungus closely resembling Phytophthora erythroseptica Pethybridge was readily obtained and the disease was diagnosed as "pink rot," first described in Ireland in 1913 by G. H. Pethybridge.

A low portion of the field from which these particular potatoes came was under water for several days during a rainy spell in September. Over 50 percent of the tubers from this area were left in the field because of the rot, and about 10 percent of those stored were diseased less than one month after digging.

This is apparently the first record of pink rot in New York State, although it has been suspected in recent years of being present to a limited extent.

The disease has been generally associated with excessively high moisture conditions in the few States in this country from which it has been reported. It is not believed that pink rot will be of any great consequence in New York State except for some local significance in low lying areas subjected to heavy rains or late irrigation.

EXTENSION PLANT PATHOLOGY, CORNELL UNIVERSITY, ITHACA, NEW YORK

GRAY WALL OR VASCULAR BROWNING OF TOMATOES IN PENNSYLVANIA

By L. P. Nichols and Edwin E. Honey

A disease affecting tomatoes was seen in commercial tomato fields and greenhouse plantings and in a few home gardens during the summer of 1950. Fruit affected showed translucent grayish-brown blotches or streaks on the outer surface which enabled the grower to grade them out. When a cross section was made through the translucent areas a brown necrosis of the vascular system was noted. These symptoms seemed similar to these described by Conover (P.D.R. 33: 283-284, 1949), Holmes (Phytopath. 40:13, 1950), and Cox and Weaver (Phytopath. 40: 870, 1950).

In some cases affected fruit were found only under dense foliation while in other fields there seemed to be no correlation between shade and appearance of symptoms.

Although gray wall has caused loss in previous years in Pennsylvania, growers are becoming increasingly alarmed about this trouble. During the season of 1950, 33 percent of the crop in one greenhouse and up to 25 percent of the crop in some commercial fields were affected.

DIVISION OF AGRICULTURAL EXTENSION, PENNSYLVANIA STATE COLLEGE, STATE COLLEGE

BLACK SHANK ON TOBACCO IN PENNSYLVANIA

By A. H. Bauer

Black shank on Pennsylvania broad leaf tobacco was found on a farm near New Providence, Lancaster County, Pennsylvania, during the past season. The identity of the disease was ascertained by E. E. Clayton and E. O. Schneider, U. S. Department of Agriculture, and W. S. Beach, Pennsylvania State College, in a visit to the locality on August 25, 1950.

More than 200 plants of approximately an acre field showed advanced symptoms of the disease, the mycelium of Phytophthora being present in affected stem tissues. Evidence given by the operator of the farm indicated that the disease was present in an adjacent field in 1949. Circumstances indicated further that the causal organism may have been brought to the locality upon tomato plants shipped from a southern State. The affected tobacco was along a creek which overflowed the field during flood while a planting of tomatoes had been made in fields up stream.

This is the first known occurrence of Phytophthora attacking tobacco in Pennsylvania. The causal organism is presumed to be P. parasitica var. nicotianae.

DIVISION OF AGRICULTURAL EXTENSION, PENNSYLVANIA STATE COLLEGE, STATE COLLEGE

DUTCH ELM DISEASE IN ILLINOIS

By J. C. Carter

Ceratostomella ulmi was cultured from branch samples of a diseased American elm located in a small roadside park on State Route 16, three miles east of Mattoon, Illinois. The diseased branch samples were collected from the diseased tree by Dr. L. R. Tehon and myself on August 3, 1950. Some of the samples were sent to Dr. Curtis May for culturing. Dr. May also reported obtaining Ceratostomella ulmi from the diseased material.

The diseased tree, as a suspect of Dutch elm disease, was reported to us by Dr. George Zentmyer of California. So far as we know, this is the first and only case of Dutch elm disease in Illinois to date.

After this case was found, State Nursery Inspectors scouted an area reaching from Effingham to Kankakee and eastward to the Indiana State line, following paved and other main roads. They took samples from seven trees, but none of these samples yielded the Dutch elm disease fungus when cultured.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS STATE NATURAL HISTORY SURVEY DIVISION, URBANA

DOWNY MILDEW ON ROSE IN CALIFORNIA

By C. E. Yarwood and Stephen Wilhelm

Downy mildew (Peronospora sparsa) of rose has cropped up recently in one greenhouse range in Alameda County and one in Contra Costa County, California. So far as anyone here knows it was last seen in 1929. To date, rainfall for 1950-1951 is about three times normal.

DIVISION OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4

ATTENTION PLEASE!

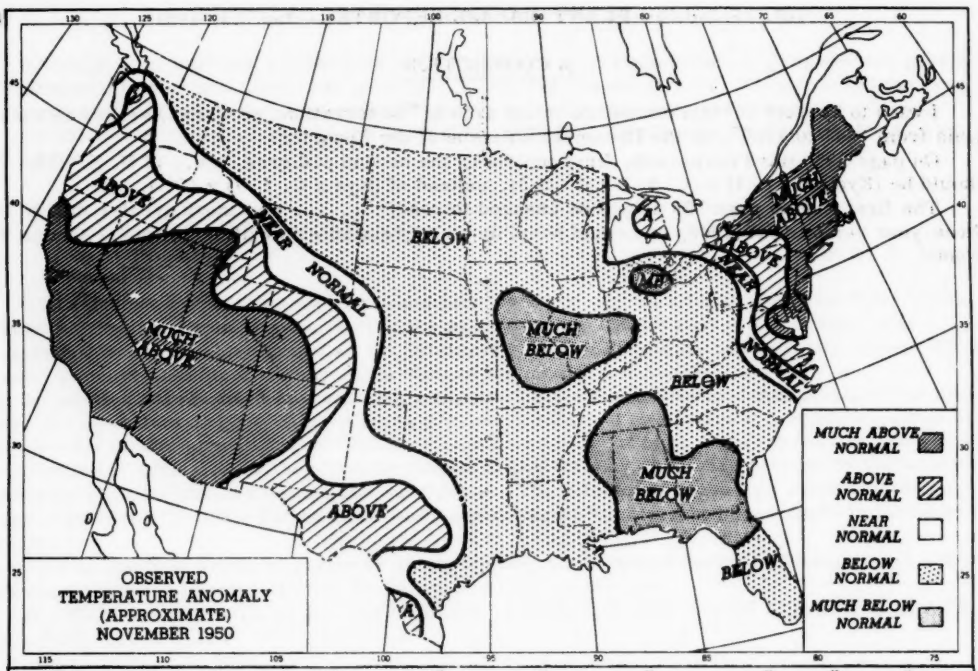
A cooperative is being organized for the purpose of exchanging stocks and information relating to Tomato Genetics. Any workers interested in joining the group should contact C. M. Rick, University of California, Davis, California.

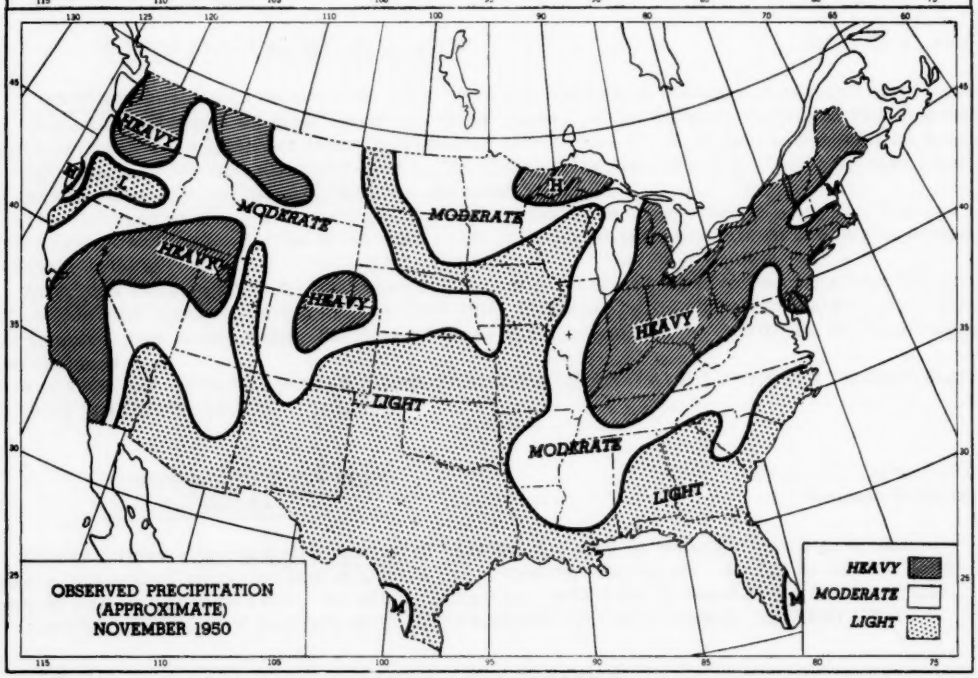
A CORRECTION

I wish to correct certain omissions in my article "Observations on Corn Diseases in Virginia from 1947 to 1950", in the December 15 issue of the Reporter.

On page 395, third paragraph, first sentence, the second pedigree, i.e., of U. S. 357, should be (Kys x C. I. 7) x (C. I. 2 x C. I. 3), instead of (Kys x C. I. 7) x (C. I. 3).

The first line on page 396 should be changed to read: "..... was much greater than in the three-year period 1947-1949, and could possibly be correlated with wilt severity." --Curtis W. Roane.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.